The New England
Journal of Medicine

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VOLUME 223 SEPTEMBER 12, 1940 NUMBER 11

CARE OF THE BACK FOLLOWING SPINAL-CORD INJURIES*

A Consideration of Bed Sores

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BOSTON

LIKE Mark Twain's weather, everybody talks about the bed sores associated with spinal-cord injuries but no one does anything about them. They are regarded as the natural accompaniment of these lesions. When a bed sore develops on a patient, the doctor in charge feels vaguely put upon, makes a variable number of futile therapeutic gestures and finally lets Nature take its course. If asked about the reasons for its development he speaks of "trophic effects" and "trophic nerves," ignoring the fact that he has not the slightest idea what either phrase means. It is my purpose to present herewith a tentative explanation of the bed-sore problem as it relates to spinal-cord injury together with the evidence on which the explanation is based and certain conclusions relative to the therapy of this annoying and serious condition.

Provided the skin is primarily undamaged, bed and pressure sores develop only because of secondary destruction of local tissue. That there are certain mechanical factors which will cause this destruction is familiar to everybody. That the latter are secondary rather than primary elements is not so well recognized, however. The occurrence and extent of this destruction depend on the presence of a bony weight-bearing prominence close beneath the skin, the thickness of the padding tissue between the bone and the skin itself, the length of time that constant weight-bearing is permitted over this point and the integrity of the protective horny layer of the skin. The first two and the last of these four factors obviously determine the usual sites at which bed sores develop. Thus, the sacrum, the trochanters, the calcanei at the insertion of the tendo Achilles,—not the heel proper,—the angles of the scapulas, the occiput and the like are usual regions where such sores may develop in a bed-ridden patient, especially if for any reason he is cachectic. The third factor is largely dependent on the presence of a local anesthetic area, and therefore becomes an integral part of the spinal-cord phase of the problem. Under ordinary circumstances, no one of these conditions causes destruction of tissue. There must be in addition a local anoxia and anemia.

A knowledge of the conditions that produce such anoxia is indispensable. It requires, moreover, an understanding of the anatomy and physiology of the cutaneous blood supply. The Suttons[1] give an excellent description of the anatomy involved. They state that the skin is supplied with blood through two horizontal plexuses. The arteries come from beneath and are largest in the regions exposed to pressure (volar, gluteal). They anastomose deep in the corium to form the arterial rete. From this, branches run to the upper third of the corium to form a second plexus, which is a sub-papillary network. Arterioles, certain of which anastomose with the veins beneath by way of the Sucquet–Hoyer canals, arise from this network to supply the papillary layer, hair follicles and sebaceous glands; they are end arteries. Capillaries from the arterioles enter the papillae and form beds. The walls of these capillaries have tonus because of the presence of the cells of Rouget. The normal blood pressure of skin capillaries approximates 20 to 40 mm. of mercury, but the walls can withstand an expanding pressure of 100 mm. The normal pressure rises to 35 to 45 mm. in reactive hyperemia and to 70 to 80 mm. in response to the local application of heat. The epidermis has no blood supply. Anatomically, then, it is apparent that the papillae, lying immediately below the avascular epidermis, contain contractile capillaries arising from arterioles

*Read in part before the joint meeting of the Philadelphia Academy of Surgery and the Boston Surgical Society, Incorporated, on March 4, 1940, at the Boston City Hospital, Boston.

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which in their turn are the end vessels of two deeper plexuses. There is a possible direct communication between the arterioles and the veins. The veins otherwise approximate the arrangement of the arteries and are also contractile.

Much of the physiology of the blood supply to the skin, especially in parts other than the extremities, is still either controversial or unknown. The mass of conflicting and confusing literature has been reviewed and oriented up to 1939 by McDowall[2] in his volume, *The Control of the Circulation of the Blood,* which supplements the earlier and more limited work of Kuntz.[3] I quote from both references freely. Certain tests have been shown to produce constant reactions. For example, stroking and local compression of the skin, under normal conditions, produce first an area of pallor, which is due to a localized constriction of the capillaries. The reaction takes place in regions in which nerves have degenerated or have been anesthetized as well as in regions with the nerve supply intact. After a definite interval and after the pressure has been removed, the pallor is ordinarily succeeded by an irregular-shaped blush or flare. The flare depends for its presence on the integrity of the peripheral nerve supply to the injured area and is due to arteriolar dilatation. A more prolonged pressure produces local tissue asphyxia which may have wheals or blisters associated with it. This is known as reactive hyperemia, and is a type of vasodilatation produced by the presence of an abnormal amount of local metabolic substances. It follows only such stoppage of the local circulation as causes tissue damage. In addition, prolonged pressure on the skin causes first pain and then local anesthesia. It is this local reaction that produces pressure sores as distinct from bed sores. As will be seen, the latter develop when associated with a spinal-cord injury, after certain other changes have been added to this fundamental reaction.

Except for this purely local mechanism, which as noted above is dependent on the integrity of the regional nerve supply, the physiological control of the cutaneous blood supply is believed to rest in the main on reflexes that are mediated by way of the sympathetic and parasympathetic nervous systems. The sympathetic pathways are well known so far as the skin is concerned, the impulses that pass over them being exclusively vasoconstrictor in type. The reflex arc lies entirely below the level of the sensory thalamus,[4] and can be shortened sufficiently to be limited to the spinal cord. The parasympathetic pathways for impulses to the vessels in the skin are unknown. There is some evidence that the latter leave the spinal cord by way of the posterior spinal roots and reach the cutaneous vessels by way of the peripheral mixed nerves. In all probability, this parasympathetic reflex arc can also lie within the limits of the spinal cord, but its connections are not certainly known. The impulses are vasodilator in type. Normally, according to Bozler,[5] it may be assumed that stimulation of the dorsal spinal roots produces a vasodilator substance at the nerve endings and stimulation of the sympathetic system a vasoconstrictor substance. If these two substances are not in balance and mutually neutralized, either vasoconstriction or vasodilatation will follow.

Thus it may safely be said that under normal conditions an adequate supply of blood to the skin depends on the integrity of two reflex arcs, which must include at least the spinal cord. One

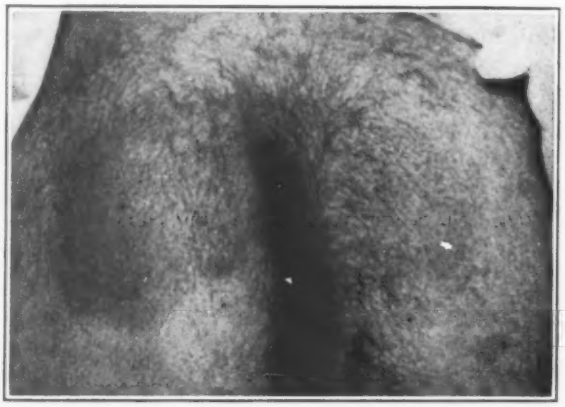

FIGURE 1. *Hyperemia over the Sacral Region.*
In this patient, who had a cervical cord injury, part of the hyperemic area necrosed later to become a pressure sore.

of these — the sympathetic — is well understood and has central connections in man in at least the thoracic and upper lumbar cord. The pathways of the other are not well known but probably include the dorsal spinal roots. Proper response on the part of the cutaneous circulation to varying local needs requires instant correlation between these two reflex mechanisms. Failure on the part of either necessarily impairs the vitality of the cutaneous tissue that their interactivity is designed to protect. In addition, local injury to the skin produces strictly local changes in the blood supply. These are in the nature of a reactive hyperemia, which may be associated with tissue asphyxia but which does occur independently of the central reflex connections. Failure of the reflex control of the cutaneous circulation, when added to the local changes that have produced or will produce a pressure sore, causes a bed sore to develop on this site.

Whether or not the spinal cord is involved, one may assume that any of the usual conditions is able to produce a pressure sore. Such a sore is generally noted first as a localized area of redness (Fig. 1). The epidermis then disappears, either by way of a blister or on the dressing, without any other noticeable preliminary, and the reddened area is changed into a weeping surface on which the papillae are clearly visible (Fig. 2). Further progress usually depends on the effects of local infection. If, however, the ulcer is kept sterile, and the causative factor is allowed to persist, deeper necrosis associated with serous discharge and further marked hyperemia follows, after which local infection is inevitable. With the onset of infection, the ulcer becomes a classic septic, granulating wound containing varying amounts of necrotic tissue (Figs. 3 and 4). Its

FIGURE 2. *Necrosis and Loss of Epidermis.*
Note the prominent papillae and the appearance of exuding serum on the surface.

progress depends on the adequacy of the surgical care. One characteristic stands out, however. Except in patients with extreme cachexia or in the presence of unreasonably prolonged immobility, the pressure sore has little or no tendency to spread radially, and if it does so, the reason can always be found in the inadequacy of the surgical treatment or in the failure to correct the underlying causative factor. Common examples of the latter are the unwillingness of the surgeon to remove a cast that is causing local pressure, and of the former the ignorance on the part of all attendants of the indispensability of a constantly dry bed, as well as their lack of knowledge as to how to assure it. Such pressure sores develop despite the normal activity of the sympathetic and parasympathetic reflexes. The first stage, which is ordinarily

FIGURE 3. *A Pressure Sore with Necrosis.*
This had failed to enlarge after being present for nine months. The patient had a transection of the cervical cord.

not seen, is pallor of the skin from local anemia. This is followed by the characteristic flare. It exemplifies the classic reactive hyperemia, with

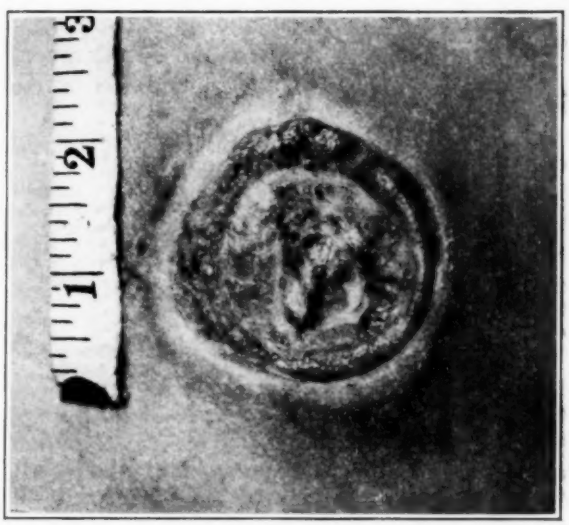

FIGURE 4. *A Pressure Sore with Local Infection.*
This was the same age and on the same patient as the one illustrated in Figure 3. The location was different, however.

local tissue asphyxia and all the accompanying chemical and cytologic changes. Such pressure sores can be caused by any of the mechanical factors noted above.

On the other hand, bed sores that develop in association with spinal-cord injuries are different from pressure sores. They start earlier in the course of the disease, spread radially almost at

FIGURE 5. *A Typical Bed Sore.*

Note the tendency to radial spreading. This patient had a transection of the thoracic cord. At the time the bed sore was photographed, it had been present for six months.

once (Fig. 5), and continue to do so for long periods. They may resume their lethal spread at any time after it has been stopped if the patient becomes exhausted or develops any significant infectious process. All this is in addition to the reactive hyperemia and local anoxia of the underlying pressure sore.

Based on our knowledge of the deleterious effect of spinal shock on all reflex activity below the level of any spinal-cord injury, its presence immediately after the injury and its characteristic continuation or recurrence whenever exhaustion or sepsis develops, I have assumed that the difference between the ordinary pressure sore and the bed sore associated with any injury to the spinal cord exists because of the influence of spinal shock on the peripheral vasomotor reflexes. An analogy is found in the effects of spinal shock on the bladder, the mechanism of which is now well established.[6] Reasoning from this analogy as well as from theoretical grounds, backed by such knowledge of the vasomotor reflexes as is possessed, bed sores should be especially common in thoracic-cord and upper-lumbar-cord lesions; that is, with the bone injury between the first dorsal and the first lumbar vertebra. It is here that the central connections of the sympathetic nervous system are made. As will be seen my experience bears out this theory.

During the ten years that the Neurosurgical Service has been established at the Boston City Hospital, 126 patients with spinal-cord injuries have passed through my hands. Thirty (24 per cent) developed bed sores.

There were 26 patients with thoracolumbar-cord injuries, none of whom died within the first twenty-four hours after injury, and of these only 14 (54 per cent) developed bed sores, although all may be considered to have been eligible (Table 1). The sores were all primarily sacral in location. One developed following the application of a plaster cast sixty-nine days after injury; another six months after injury, after the patient had been allowed to lie in a wet bed for the first time. Two patients were admitted with large sacral sores already present; both were said to have appeared immediately after the accidents (shootings), which occurred thirty-one days and two years previously. Of the remaining 10 patients, 1 is known to have had a bed sore develop in two hours after injury, 1 in twenty-four hours, 1 in three days, 1 in four days, 1 in five days, 3 in six days, 1 in seven days and 1 in eleven days after the accident. Nine of the 14 patients had transected cords and the other 5 suffered from contusion with a physiologic transection. One patient is still in the hospital, 1 is leading a satisfactory wheel-chair life at home five years after injury, 1 died of pneumonia at another public institution three and a half years after injury, and 1 died six weeks after returning home in a hopeless condition. The other 10 patients (71 per cent) all died in the hospital, but I am not certain as to the cause of death. I had believed it was sepsis, but recent experience leads me to ascribe the

TABLE 1. *Data on Cases with Thoracolumbar-Cord Injuries.*

LOCATION OF LESION	NO. OF CASES	CASES WITH BED SORES %	DEATHS HOSPITAL WITHIN 24 HR.	DEATHS TOTAL	HOSPITAL MORTALITY %	DEATHS AFTER DISCHARGE	TOTAL MORTALITY %	NO. OF CASES AT HOME OR AT HOSPITAL	NO. OF CASES LOST
Thoracolumbar cord									
All cases	26		0	17	65	3	77	6	0
Those with bed sores	14	54	0	10	71	2	86	2	0

deaths to more fundamental changes in the body chemistry. In support of this theory there was a marked drop in the serum-protein levels in the cases with significant bed sores but no conclusions are justifiable as to the significance of this observation. Furthermore, this drop seemed to be commensurate with the diameter and depth of the ulcerated areas.

Seventy-six of the cord injuries were cervical (Table 2). Ten patients died within twenty-four hours after their injury and must therefore be discarded as not eligible for bed sores. Of the remaining 66 patients 13 (or 20 per cent) developed bed sores. One had a sore on the occiput because of improper traction twenty-five days after the accident, 2 developed sacral sores nine and twenty-four days respectively after the accident and immediately following the application of plaster jackets, and 1 developed a sacral sore eighty-three days following the accident after being allowed to lie in a wet bed for the first time. Of the remaining 9 patients, 1 had pressure sores rather than bed sores but is included in the series nevertheless. He is still in the hospital and has a transverse myelitis from a thrombosis which followed cervical hematomyelia and edema (Figs. 3 and 4). Two of his sores are trochanteric and developed at the site of abrasions twenty-four hours after the accident. So far as the rest of these patients are concerned, the time of onset of 1 bed sore is unknown, 1 developed in one day, and 1 each within two, seven, nine, ten, fifteen and sixteen days following the injury. Seven or a little more than half of the cervical patients with bed sores died in the hospital. Two had transections, 2 had contusions, and the other 3 had hematomyelia. The cause of death was sepsis from an undrained periurethral abscess in 1 case, pulmonary embolus from a femoral phlebitis in 1 and was undetermined in the 5 others. All the 5 patients who left the hospital alive had hematomyelia. One of them died of cancer of the rectum after seven years in another public institution. The other four were self-supporting and well two, two, four, and an unknown number of years after the injury, respectively.

Twenty-four patients had injuries of the cauda equina (Table 2). None died within twenty-four hours of the injury, so that all were eligible for bed sores. Three patients (12 per cent) developed bed sores. One sore was in the sacral region, and developed thirteen days after a laminectomy accompanied by a considerable manipulation of the cauda. The operation was performed thirteen years after the injury. Another was in the lower thoracic region, and followed rapid hyperextension and the application of a plaster cast sixty days after the accident. The third sore occurred on the buttocks at the site of abrasions sustained at the time of the accident; it was present on admission. This patient died one month after admission from septicemia caused by an infected operative wound. Of the other 2 patients, one was well ten years after discharge and the other has been lost sight of.

TABLE 2. *Data on Cases with Cervical-Cord and Cauda-Equina Lesions.*

LOCATION OF LESION	NO. OF CASES	CASES WITH BED SORES %	HOSPITAL DEATHS WITHIN 24 HR.	HOSPITAL DEATHS TOTAL	HOSPITAL MORTALITY %	DEATHS AFTER DISCHARGE	TOTAL MORTALITY %	NO. OF CASES AT HOME OR AT HOSPITAL	NO. OF CASES LOST
Cervical cord									
All cases	76		10	36	47	4	53	32	4
Those with bed sores	13	20*	0	7	54	1	61	5	0
Cauda equina									
All cases	24		0	2	8	0	8	18	4
Those with bed sores	3	12	0	1	33	0	33	4	1
Cervical cord and cauda equina									
All cases	100		10	38	38	4	42	50	8
Those with bed sores	16	18*	0	8	50	1	56	6	1

*Exclusive of patients who died in the hospital within twenty-four hours.

Thus it will be seen that 54 per cent of the patients with thoracolumbar-cord injuries developed bed sores. The severity of the injury and of its complications is indicated by a group hospital mortality of 71 per cent. To be sure, practically all these patients had anatomic or physiologic transections, but this cannot be held to be the determining factor in the production of the bed sores, since such sores have developed also in association with other types of cord injury. However it unquestionably plays a part. In contrast only 18 per cent of the patients with injuries of the cervical cord and cauda equina had such bed sores. The hospital mortality of this group was only 50 per cent. These discrepancies are too great to be either coincidental or due to factors other than that of the location of the cord injury.

Of the other factors that might influence the occurrence of bed sores, the only important one

is the variation in the treatment of the cord injury as applied to the different sites of cord damage. Within the limits of the individual characteristics inherent in the injuries themselves, all, at no matter what level, have received identical treatment. At the time of admission, any surgical shock that may be present is dealt with first. Simultaneously, the patient is put on tidal drainage.[6] After the surgical shock has been cared for, a cystometrogram[7] is made and the height of the siphon curve on the tidal drainage apparatus is adjusted accordingly. A lumbar puncture is next done, and the presence or absence of a spinal subarachnoid block is determined. If there is a block and the lesion is thoracic, a decompressive laminectomy is performed unless a major dislocation is present. If it is, the laminectomy is deferred until after a moderate degree of hyperextension of the spine has been attained, by placing opposite the kyphos an increasingly enlarged roll of blankets between the mattress and bedboards which rest on the springs (Fig. 6). If the block is decreasing after forty-eight hours of this therapy, operation is usually unnecessary. Cervical cord injuries that have a cerebrospinal-fluid block are not operated on for at least five to seven days.[8] Injuries about the cauda, if acute, are treated by frequent lumbar drainage, which is continued until the fluid is free of blood. The late ones are explored. All patients are kept on the regular hospital mattress, with the bed made up in the ordinary way. Forcible rapid extension, Bradford frames, plaster-of-Paris jackets, rubber or other types of rings, lamb's wool pads and the like are not only no longer used, but are considered to be contraindicated and to be prolific sources of bed sores. The patients are kept scrupulously dry and are turned on an hourly schedule, their backs being rubbed, dried and powdered at each turning. It is interesting that profuse sweating has had no deleterious effect on the skin.

If the early signs of the presence of a pressure sore appear, the hyperemic areas are painted with tincture of benzoin twice in twenty-four hours. If the pressure sore develops into a bed sore or if the tissue destruction is merely the local type that goes with a pressure sore, the skin edge around the ulcers is treated in the same way. Sloughs and gangrenous tissue are never cut off but are allowed to stay in place until they fall off. Abscesses are tapped and emptied through the needle. Incision and drainage is contraindicated. Attempts to isolate the infecting organism have nearly always been unsuccessful on account of the large number of contaminants. However, ulcerated areas that are infected with *Streptococcus hemolyticus,* as determined by culture, are dressed with gauze saturated in a solution of sulfanilamide. Zinc peroxide as advised by Meleney and Harvey[9] should be used if the infection can be shown to be a micro-

FIGURE 6. *Hyperextension of the Thoracic Spine by Means of a Blanket Roll and Bedboards.*

aerophilic hemolytic streptococcus. No such case was recognized in this series. Other ulcerated surfaces are wiped clean twice in twenty-four hours. No local application is used. High-vitamin and latterly high-protein diets, transfusions as indicated, adequate fluids, appropriate chemical treatment of the bacteriuria that is always present and physiotherapy are adjunct measures. In the past, I have tried and discarded all the ordinary forms of local applications, including tannic acid, gentian violet, scarlet red, Enzymol and so forth, strapping of the ulcer as recommended by Carty,[10] all types of ring supports under back and heels, Bradford frames, sawdust beds and lamb's wool pads. The same may be said of all types of mattresses except the modern, sponge-rubber one, which promises to be helpful.

My experience thus indicates that the only therapeutic essentials to prevent the development of bed sores are to move these patients on an exact hourly time schedule; to avoid, except as described above, all forms of external artificial splinting or support to the spinal column so long as the patient is bedridden; to prevent the development of any serious sepsis or exhaustion; and to keep the patient constantly and completely dry — a desideratum that can be accomplished only by the use of a properly adjusted tidal-drainage apparatus. Tincture of benzoin is the only worthwhile local application, and sponge-rubber mattresses appear to be desirable and useful in thoracic-cord injuries. Active surgery and wet dressings are contraindicated.

Discussion

The occurrence of both bed and pressure sores should be explainable by one fundamental process. This process should be based, if possible, on known facts, and should eliminate such highly theoretical concepts as are suggested by the unexplainable terms "trophic nerves" and "trophic changes." Although some gaps remain to be filled in, it will suffice for the present to ascribe both types of lesions to a local tissue necrosis which is caused by a local impairment of the circulation. The therapeutic importance of this concept is obvious.

The application of the well understood experimental changes which, in a mild form produce local anemia and the hyperemic flare, to the more advanced clinical condition recognized as a pressure sore and accompanied by a variable degree of such local necrosis, seems well supported by known facts. That this is the first stage of the more complicated bed sore characteristic of spinal-cord injuries appears equally reasonable. Experimental evidence and certain physiological and clinical data in support of the latter contention have, however, been lacking. The demonstration herewith presented of the preponderance and greater seriousness of bed sores associated with thoracic as contrasted with all other cord injuries provides a great deal of the missing clinical proof. This is particularly true when the importance of the reflex control of the cutaneous circulation and of its intimate association with the thoracic cord by way of the central connections of the sympathetic nervous system is appreciated. If the immediate effect of the presence of spinal shock on this as well as all other reflex activities below the level of a cord injury, and its characteristic later recurrence in the presence of a serious infection or exhaustion, are added, the process, while not proved in detail, is so clear as to supersede any other more theoretical explanation.

From a practical point of view, such an explanation is of great significance. To be sure, without further evidence it can apply only to the bed and pressure sores that develop in association with spinal-cord injuries or other processes — such as an operation — known to produce spinal shock. Furthermore, care must be taken not to confuse the causative factors outlined in detail in this paper with the conditions, such as infection and the like, that do not cause but rather prolong the life of bed and pressure sores after they have developed. This situation has barely been touched on. If this explanation is accepted and properly applied, it commits the surgeon to a form of preventive therapy that is primarily concerned with conserving and improving the local circulation. His attention must therefore be focused on those procedures that eliminate, so far as possible, prolonged local weight-bearing or irritation, that prevent all maceration of the skin and the development of any major systemic infectious process and that otherwise tend to eliminate spinal shock or prevent its recurrence. Locally, he will avoid such applications and surgery as are in the least degree destructive. He will recognize also that although bed sores may be associated with any level of injury to the spinal cord and cauda equina, they are three times as common and one and a half times as fatal in thoracic-cord injuries as they are in other spinal-cord injuries. He should therefore be more than usually careful in such cases to bear in mind the need for conserving their cutaneous circulation.

Summary and Conclusions

An explanation of the occurrence of pressure sores is offered. This is based on the presence of the well-recognized experimental cutaneous hyperemic flare.

The occurrence of bed sores in association with spinal-cord injuries is explained on the theory that all bed sores start as pressure sores, but develop more viciously and more widely because of the deleterious effect of spinal shock on the local cutaneous vascular reflexes.

Clinical evidence in support of these explanations is offered by virtue of an analysis of 126 cases of spinal-cord and cauda equina injuries, in which 30 (24 per cent) of the patients developed bed sores.

Bed sores occurred in 54 per cent of 26 thoracolumbar-cord injuries. The hospital mortality in this group was 71 per cent.

Bed sores occurred in 18 per cent of the cervical-cord and cauda-equina injuries. The hospital mortality in this group was 50 per cent.

On the basis of the fundamental conception that bed sores are specialized and more virulent forms of pressure sores which are associated with spinal-cord injuries, and which are themselves merely evidences of a local cutaneous circulation that has failed to the point of producing local tissue necrosis, therapy designed to conserve and prevent further interference with the cutaneous blood supply is recommended.

The essentials of this treatment are as follows: to move such patients — and particularly those with thoracic-cord injuries — on an hourly time schedule; to prevent the development of any serious exhaustion by appropriate feeding, transfusions, administration of vitamins and the like; to prevent the occurrence of sepsis and to maintain a constantly dry bed by the proper use of a tidal-drainage apparatus; and to avoid all forms of external artificial splinting or support to the spinal column so long as the patient with a spinal-cord injury is bedridden.

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TUBERCULOSILICOSIS*

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THIS paper reports the observations made in the serial examination of 207 employees in the granite industry who had silicosis complicated by pulmonary tuberculosis, and who were working on full or part time when originally examined.

PATHOGENESIS

Tuberculosilicosis can be defined as the condition in the lungs caused by the combined action of tubercle bacilli and silica dust. It differs from tuberculosis in a non-silicotic individual in that the disease process is not due to the tubercle bacillus alone. The silica appears to impair the lymphatic drainage of the lung. Linear and nodular fibrosis results, with frequent obliteration of the pulmonary capillaries. In pneumoconiosis caused by non-siliceous dusts, linear and focal pigmentations are produced as in chronic infections and arteriosclerosis, but the discrete nodulation is not present as in silicosis. In silicosis the resulting local cellular proliferation consists primarily of nodules about 2 to 5 mm. in diameter, composed of concentric layers of fibrous tissue, which are quite similar to those formed by tubercle bacilli.[1]

It has been shown clinically, pathologically and experimentally that this characteristic fibrosis of the lung produced by silica predisposes the patient to tuberculosis. Inorganic dusts, including silicates, with the possible exception of asbestos, do not generally result in such fibrosis unless mixed with some amount of free silica.

The fibrous nodule is the characteristic lesion of silicosis, but a differential diagnosis between silicosis and tuberculosilicosis is one of degree. There are no standards for a definite opinion in the borderline cases. It is stated[2] that the amount and distribution of the collagen in the tissue reaction may help in differentiation. One notes that

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in the presence of active tuberculosis the characteristic silicotic nodules are no longer clear-cut and sharply defined, the borders become irregular, and as the disease process advances the localized dense massive areas show ill-defined radiating borders.

Silicotic nodules may be present in the early stage of the disease and the x-ray examination may yet be negative. Tuberculous infection may rapidly accentuate the nodules. In cases of silicosis preceded by short exposures to high concentrations of silica and followed by early death, microscopic sections from autopsy material have shown many silicotic lesions not revealed by the x-ray examination. Almost all these cases have a very acute tuberculous process.[3]

All observations appear to point to free silica as the substance that increases the liability to tuberculous infection. The silica is believed to act as a chemical irritant of pulmonary tissue and to be associated with the solution of the quartz found in granite. The rate of reaction is probably proportional to the size of the particles.[4] Only free silica seems to have any specific action on tubercle bacilli in tissue.[5] At the present time most of the evidence points to a decrease in tissue resistance to tubercle bacilli among patients with silicosis. Silica appears to enhance the growth of tubercle bacilli,[6] and there is ample evidence to show that tubercle bacilli grow rapidly in the presence of silicotic lesions;[7] but there is no proof that tubercle bacilli obtained from patients with tuberculosilicosis show an increased growth.[8]

It is possible that the non-siliceous components of granite dust reduce the effect of the free silica in quartz dust.[9] Clinical observations have shown that long exposures to granite dust are necessary to produce nodulation in men with no reinfection tuberculosis. There seems to be some protection against silica dust in the granite mixture, either by reduction of the total amount of inhaled quartz or by the chemical inhibition of the reaction of the silica in the body. Injection of granite dust into experimental animals has not caused so much reaction as has the same amount of quartz alone in the dust. Inhalation experiments have shown the same results.[5]

Inhalation appears to be the most frequent mode of entry of the tubercle bacilli in primary and exogenous reinfection tuberculosilicosis. Moist sputum droplets, infected dry dust and contact are the commonest methods of cross-infection.

Ever since Collis pointed out the high incidence and chronicity of tuberculous infection in silicotic patients, experience has shown that many cases of tuberculosilicosis are found if such patients are followed long enough. A quiescent, encapsulated tuberculous focus may be spread by the transportation of silica to the diseased area. The organisms may live for a long time in the silicotic mass without appearing to multiply there.

Occupational and Environmental Factors

The diagnosis of tuberculosilicosis requires, first, that the existence of a silicosis hazard shall have been proved. The occupational history and a study of the patient's environment must show the presence of dangerous concentrations of free silica in the breathing air level. This danger limit in the granite industry is usually set at 10,000,000 particles per cubic foot of air.

Petrographic analyses of granite samples from the Barre, Vermont, district have shown a content of 31 to 38 per cent of silica.[12] Average dust counts in the granite-cutting trade have ranged from approximately 60,000,000 particles per cubic foot of air for hand-pneumatic tool operators to less than 10,000,000 particles for attendant laborers.[13] The proof of silicosis usually requires an estimate of the time spent by the workman in the dust. In granite-cutting, since the exposure is quite constant, the history of the duration of the time spent in the industry is sufficient. On the other hand, the granite-quarrier, whose work is more seasonal, requires a more detailed occupational study. These men have been exposed to dust counts ranging from approximately 150,000,000 particles per cubic foot of air for the leyner drillers to 40,000,000 for the plug drillers.[13] It has also been shown that the dangerous particles are between 0.5 and 10 microns in diameter. In granite-cutting, 94 per cent of the dust particles have been found to measure less than 3 microns.[14]

Another contribution toward the diagnosis of tuberculosilicosis is the finding of a high local rate of pulmonary tuberculosis in any industry and its environment. From 1915 to 1936 the rate of actual to expected deaths from pulmonary tuberculosis among granite and sandstone stonecutters rose from 976 to 2693 per cent, in other words the actual deaths were twenty-six times those expected.[15]

The tuberculosis death rate has tended to remain high in Washington County, the center of the granite industry in Vermont, averaging 154 per 100,000 population per year for the period 1934–1937. The mortality for the rest of the state averaged 34 per 100,000 per year during the same period, or about one fifth of that of Washington County.[16]

Symptoms and Signs

The onset of tuberculous infection in silicotic patients may be accompanied by no symptoms, by a general indisposition or by an explosive widespread dissemination of the disease process. The commonest symptom is dyspnea. This term is generally reserved for the unusual breathlessness of which the patient complains following the exertion he undergoes at his regular occupation. He becomes conscious of a breathing effort that he had not noticed previously. In uncomplicated silicosis there may be a slight shortness of breath, with dyspnea and cough present later. As the tuberculous infection sets in the dyspnea usually increases, and appears altogether out of proportion to the amount of diseased area present.[18] The number of granite workers with tuberculosilicosis who show definite dyspnea appears to increase with the years of service in the industry (Table 1).

Table 1. *Percentage Distribution of Symptoms, by Years of Service.*

Length of Service in Granite Industry	Dyspnea	Cough	Systemic Symptoms
yr.	%	%	%
0 – 10	40	20	0
10 – 20	44	52	32
20 – 30	62	64	26
30 and over	83	65	10

Table 2. *Percentage Distribution of Symptoms, by Age Groups.*

Age	Dyspnea	Cough	Systemic Symptoms
yr.	%	%	%
20 – 30	0	0	100
30 – 40	30	30	12
40 – 50	66	78	37
50 – 60	62	51	16
60 and over	75	75	17

This shortness of breath is frequently very difficult to differentiate clinically from that caused by chronic pneumonitis with bronchiectasis, bronchial asthma, pulmonary fibrosis with emphysema, carcinoma of the lung and cardiovascular disease due to arteriosclerosis, hypertension or syphilis. Cough, expectoration, chest pain and hemoptysis often accompany the onset of tuberculous infection in silicosis. As in pulmonary tuberculosis without silicosis, there may be systemic symptoms of toxemia consisting of weight loss, fatigue, weakness, dyspepsia, morning nausea and vomiting, nervousness, night sweats and fever. These seem to appear most frequently in granite workers at about the age of forty-five (Table 2).

The physical examination in cases of tuberculosilicosis is extremely variable. Most cases with early infection are free of physical signs. With consolidation and fibrosis present and predominant, there may be found diminished expansion, impaired resonance, diminished breath sounds and rales. Sibilant rales may be the only signs of conglomerate fibrotic areas. Cavities may result in bronchial or amphoric breathing. The correlation of the symptoms and signs through the medium of the physical examination frequently determines the degree of disability of the worker.

Roentgenological Findings

Bilateral nodular shadows in the chest x-ray films of granite workers are usually due to simple silicosis. However, it is difficult through the medium of a single film to differentiate early uncomplicated silicosis and tuberculosis. Silicotic nodules are frequently seen first as discrete nodular areas in the midportion of the lung fields. Their coalescence is represented by massive shadows. In uncomplicated silicosis the hilar shadows often recede even though the lung fields show more intense nodulation. Tuberculous infection is often noted in those cases in which the hilar shadows enlarge and become sharply defined. The onset of tuberculous infection is also noted in chests showing the appearance of hazy infraclavicular areas on one side during an interval of a few months. The nodular shadows present in previous films now rapidly grow irregular in shape and distribution.

Table 3. *Results of Direct Smear Examinations of Sputum.*

Age	No. of Cases	No. of Patients Permitting Sputum Examination	Percentage of Cases with Tubercle Bacilli in Sputum
yr.			%
20 – 30	1	1	100
30 – 40	17	7	43
40 – 50	55	16	69
50 – 60	82	21	76
60 and over	52	15	33

Table 4. *Relation Between X-ray Evidence of Cavitation and the Finding of Tubercle Bacilli by Direct Smear in the Sputum.*

X-Ray Diagnosis	No. of Cases	Percentage of Patients Permitting Sputum Examination	Percentage of Cases with Positive Sputums
		%	%
Silicosis with infection and cavitation	118	40	71
Silicosis with infection and no cavitation..	89	13	25

In tuberculosilicosis the linear apical shadows

that may be present seem to be broader than those noted in cases of pulmonary tuberculosis without silicosis. Unless these are in the top lung fields, tuberculosis with silicosis is difficult to differentiate from conglomerate silicosis. But one notes that the conglomerate nodular areas persist unchanged for several years, whereas with infection present the edges of the nodules become fluffy and ill-defined.

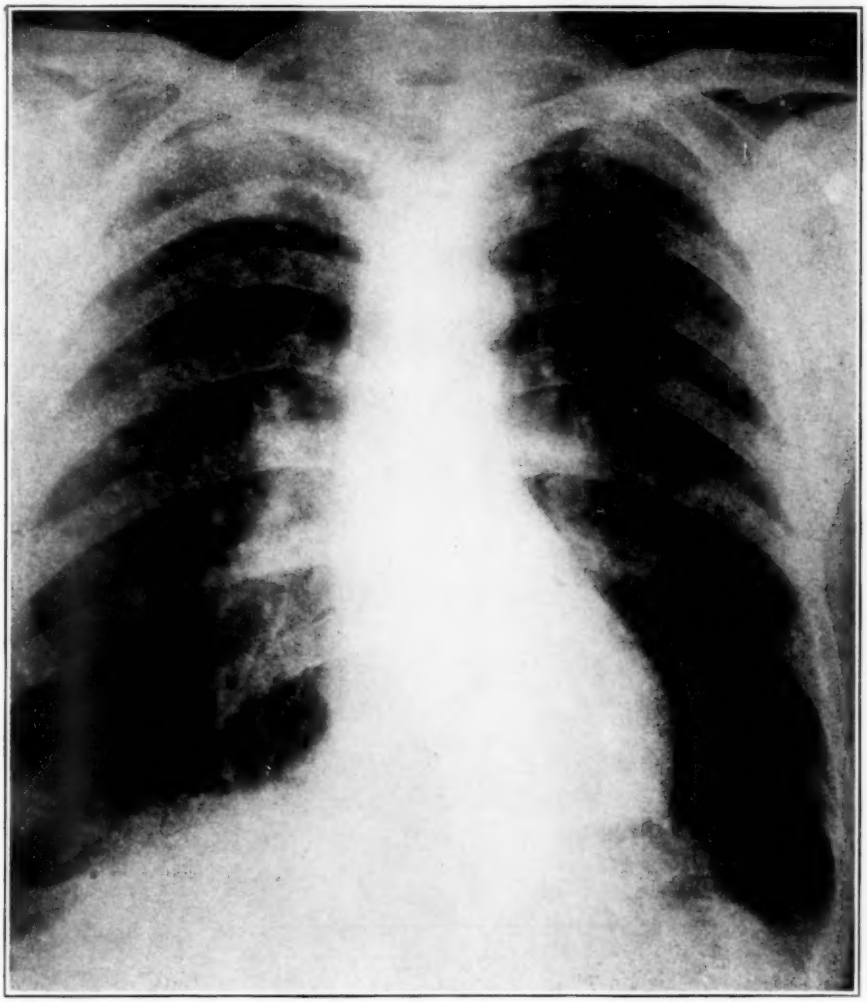

FIGURE 1.

X-ray photograph of the chest of a hand-pneumatic-tool operator in granite for seventeen years, showing bilateral discrete nodular shadows with well-defined borders, and localized density in the right upper lobe.

This man was forty-five years old and had no symptoms or signs at the time of the original examination. Serial examinations during a five-year period have shown no change.

This case is one of silicosis with inactive tuberculosis.

LABORATORY FINDINGS

The only absolute clinical proof of tuberculosis is the discovery of tubercle bacilli in the sputum. Direct smears usually detect tubercle bacilli only when the sputum contains at least 100,000 organisms per cubic centimeter. Consequently, frequent concentration and animal-injection examinations are required for proof of the presence of the bacilli. But even direct smears will show a high percentage of positive sputums in forty- to sixty-year-old patients with tuberculosilicosis (Table 3).

There appears to be a high correlation between the x-ray findings of cavities in tuberculosilicosis and tubercle bacilli in the sputum (Table 4).

The sedimentation rate is of some prognostic value when used serially. Not infrequently one notes normal sedimentation rates in cases of tuber-

culosilicosis that are progressive and have positive sputums.

A negative tuberculin test aids in the rare case as a differential guide.

Clinical Forms

The clinical types of tuberculosilicosis are often similar to those seen in pulmonary tuberculosis without silicosis. The primary complex already present is seldom influenced by the inhalation of silica. The lesions are usually healed. They can be reactivated but only rarely. On the other hand, primary infection can occur in patients with silicosis a few weeks after the inhalation of tubercle bacilli. Frequent infection with tubercle bacilli in these patients may lead to reactivation, caseation or dissemination of the process.

The commonest type of tuberculosilicosis seen in granite workers is that in which there is a very slow progression of the infectious process. Many of these cases appear in patients who have previously shown localized discrete areas by x-ray accompanying nodular shadows of simple silicosis, and suggesting the occurrence of silicosis superimposed on inactive tuberculosis (Fig. 1). The chronic course seems to follow the reactivation of the old lesions, and begins chiefly in the upper lobes and extends to the lower fields. The consolidation increases yearly, and basal infiltration is quite common. There are cough, dyspnea,

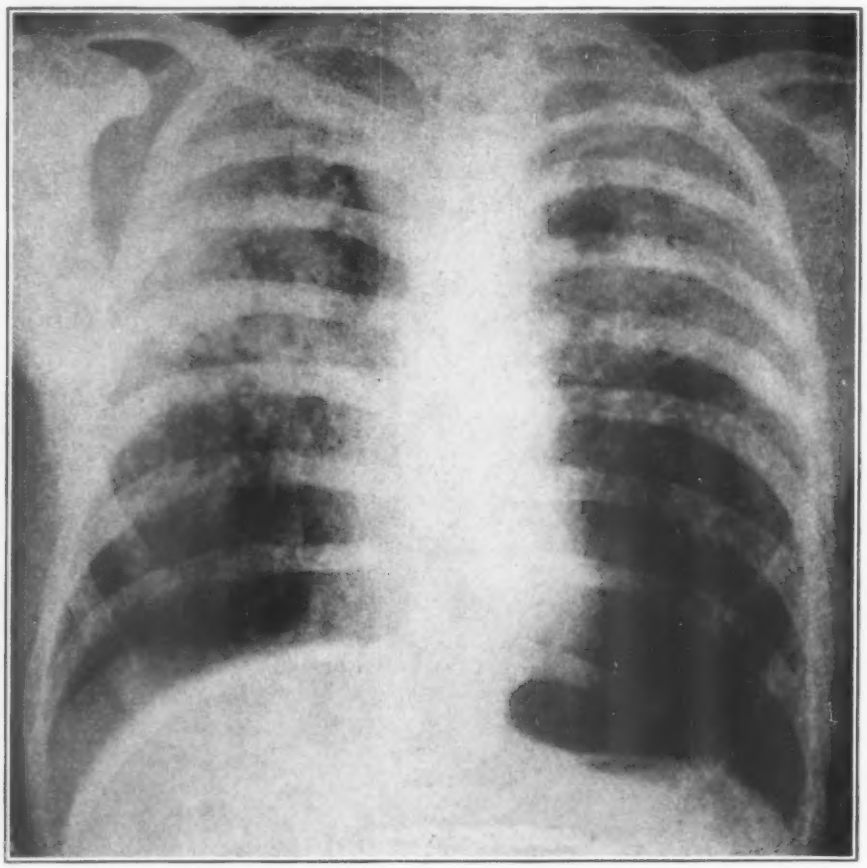

Figure 2.

X-ray photograph of the chest of a man working as a hand-pneumatic-tool operator in granite for fourteen years, showing massive shadows of homogeneous density asymmetrically distributed over areas of mottled nodulation with regions of increased transparency.

At the original examination this man complained of cough for six years, increasing dyspnea for four years, night sweats for two years and a 15-pound weight loss in two years; examination disclosed bilateral dullness and crepitant rales, a sedimentation rate of 18 mm. in one hour and, very rarely, a positive sputum.

He entered the sanatorium three times in four years, each time returning to work after two or three months. He was up and about, though moderately dyspneic, six days before death. There was x-ray evidence of cardiac enlargement two months before death.

distorted thorax and later purulent expectoration. The patients are usually afebrile but have toxic symptoms off and on. The sputum is ordinarily negative until the terminal months, but may be positive intermittently. The x-ray examination may reveal massive consolidation on conglomerate fibrotic areas. Later there is peripheral extension and cavity formation, which can best be seen in many by overexposure of the film (Fig. 2). The cavities may become invisible to the x-ray and a period of quiescence may follow. Usually progressive fibrosis, cavity formation and a final pneumonic spread complete the story. Some of these patients die with evidence of cardiac dilatation by x-ray, preceded by venous engorgement and ascites.

A less frequent type seen is that which takes a comparatively rapid course with very early cavitation. The onset is sudden, with all the toxic symptoms of an acute bronchopneumonia, such as productive cough, dyspnea, fever, hemoptysis and rapid weight loss. Serial examinations before and after the onset of acute symptoms suggest that this form of tuberculosilicosis frequently fol-

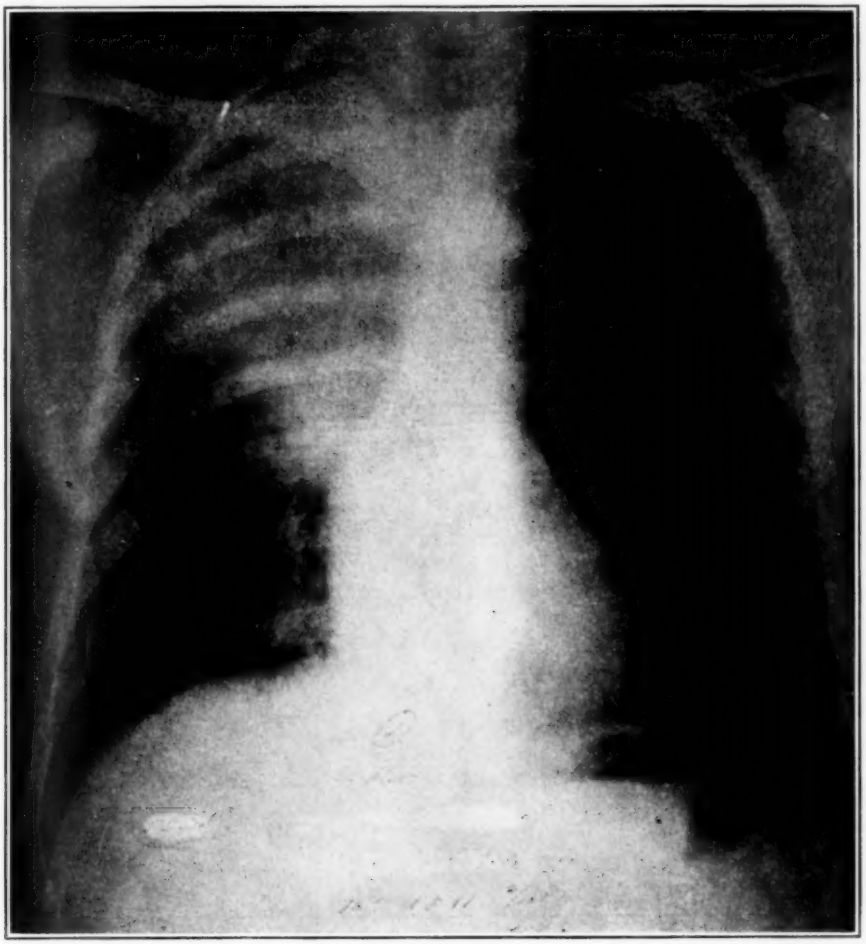

FIGURE 3.

X-ray photograph of the chest of a man who had worked as a hand-pneumatic-tool operator in granite for thirty-eight years, showing massive homogeneous density in the right upper lung and cavitation and ill-defined nodular shadows in both lungs, with greater mottling on the right and increased transparency and loss of fine detail on the left.

This man was entirely well up to one month before the first examination, when there was sudden onset of cough, expectoration, dyspnea, weakness, nervousness and right chest pain, with a 16-pound weight loss in one month. Dullness and crepitant rales were found on the right. The sputum was positive, and the sedimentation rate was 24 mm. in one hour. At this time he was fifty-seven years old.

The symptoms increased with bed rest.

lows a recent tuberculous infection in a long-standing case of silicosis. The silicosis appears to exert very little influence on the course of the infection, which seems to spread by aspiration as well as by extension. Physical signs are almost always present, and are those of consolidation and cavitation. The x-ray examination discloses mottling on a nodular background, greatest in the upper lobes, and the presence of rapidly progressive cavitation. The sputum in these cases is markedly positive from the onset of the acute symptoms. The patient rapidly becomes worse with all the symptoms and signs of toxicity, and dies of sepsis (Figs. 3 and 4).

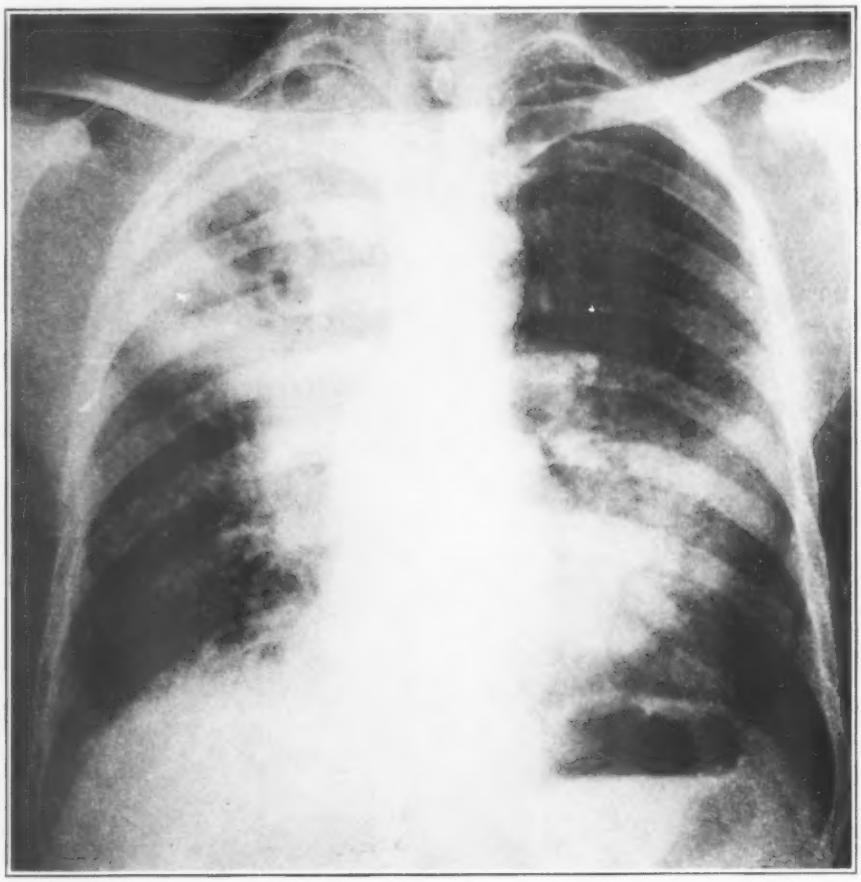

FIGURE 4.

X-ray photograph of the chest of the man shown in Figure 3, taken four months later and showing increase in the size and number of the cavities on the right, with irregular distribution of soft mottling in the left lower chest.

At this time the patient was markedly dyspneic on the slightest exertion. He died approximately eight months after the onset of the acute symptoms. Autopsy revealed bilateral silicotic nodules, with tuberculous cavitation, in the right upper and lower lobes. In the left lung the alveoli about the silicotic nodules were filled with exudate. Tubercle bacilli were demonstrated in the necrotic areas, almost wholly among the polymorphonuclear leukocytes; none were seen in the silicotic nodules or in the surrounding tissue.

A still less frequent form of tuberculosilicosis noted in granite workers is that which sometimes occurs among young men. The onset is acute, with marked dyspnea, cough and fever, but there is very little expectoration or loss in weight. Recent x-ray films of the chests of these patients show the nodular shadows of simple silicosis. Films taken immediately after the onset of symptoms disclose marked fuzzy borders about the nodules, which increase markedly in size within a few months. There also appears to be rapid coalescence of the nodular shadows. Physical findings at the onset are almost always negative, and one can rarely demonstrate any tubercle bacilli in the sputum. The acute process in these cases may become chronic. This form of tuberculo-

silicosis may be the result of a primary or lymphohematogenous infection in a patient with silicosis and very little resistance to tuberculosis (Fig. 5).

Prognosis

Patients with silicosis are not certain to develop tuberculosis. Cases of silicosis with early tuberculosis may improve under treatment. The extent and type of the infection regulate the prognosis. In many cases this depends on the amount of previous contact with tuberculosis. The outlook becomes poor with the presence of progressively increasing respiratory symptoms. The continued presence of tubercle bacilli in the worker's sputum carries with it a bad prognosis.[20] Of the 207 granite workers with tuberculosilicosis studied, 20 per cent died within two years after their first examination, at which time they were working on full or part time (Table 5). Approximately 87 per cent of those who died and had permitted their sputum to be examined showed tubercle bacilli on direct smear (Table 6). Concomitant complications, such as diabetes mellitus, syphilis and alcoholism, appear to increase the rapidity of the progress of the disease.

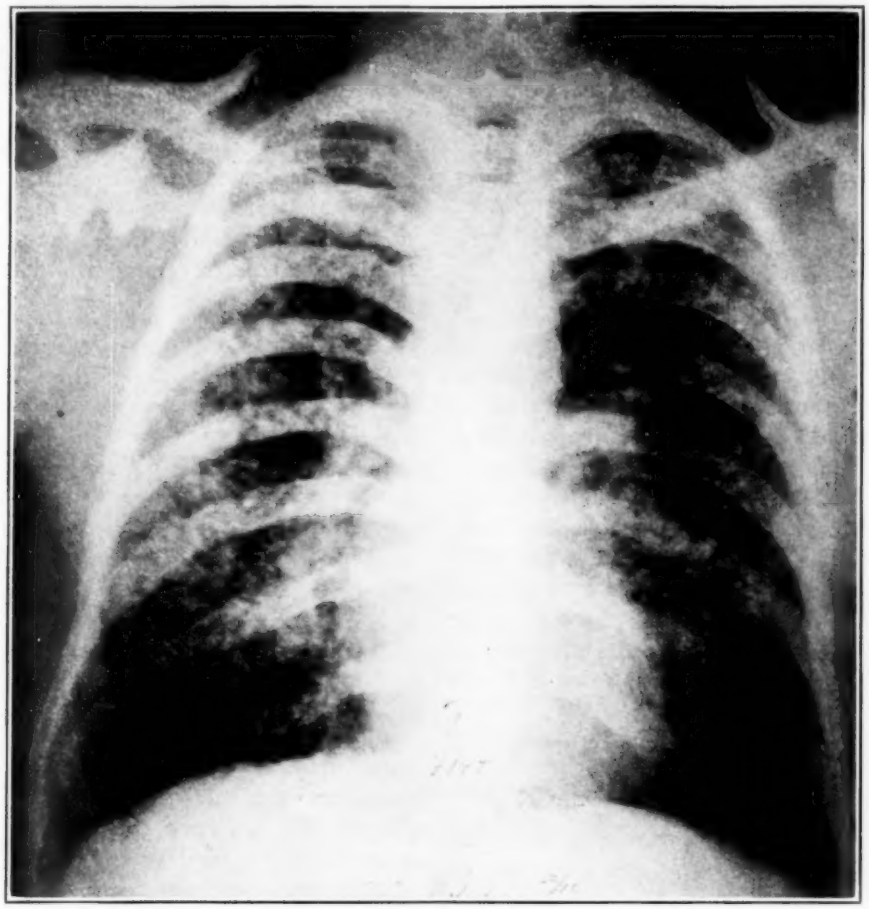

Figure 5.

X-ray photograph of the chest of a man who had worked as a surface cutter in granite for eighteen years, showing a hazy mottling about dense nodular shadows throughout both lungs, with well-defined hilar shadows.

This thirty-five-year-old man had been examined routinely eight months previously, with findings consistent with early silicosis. The tuberculin test (0.1 mg. O.T.) was negative.

The present examination was immediately preceded (within approximately five days) by the sudden onset of cough, expectoration, marked dyspnea, hoarseness and a 2°F. temperature rise in the evening. The sedimentation rate was 30 mm. in one hour, the sputum was negative, and the tuberculin test (0.1 mg. O.T.) was positive.

On bed rest, the dyspnea almost disappeared, but the temperature rise persisted.

Prophylaxis and Treatment

The prophylaxis of tuberculosilicosis consists in the prevention of silicosis and tuberculosis. The prevention of silicosis by the reduction of silica dust in the working environment has proved practical. Pre-employment examinations to detect respiratory disease before the worker can be exposed to silica dust are of value. Regular periodic examinations lead to the detection of pulmonary infection as early as possible. The removal of all cases with active pulmonary tuberculosis from contact with their fellow-workers can reduce the incidence of tuberculosilicosis.

Table 5. *Two-year Progress of Patients with Tuberculosilicosis Working on Full or Part Time at First Examination.*

Term of Service in Granite Industry	No. of Cases	Deaths Within 2 Yr. After Examination
yr.		%
0 – 10	5	0
10 – 20	25	28
20 – 30	61	21
30 and over	116	18
Total	207	
General average		20

It must be remembered that a laborer with tuberculosilicosis and a positive sputum can infect not only his fellow-workers but his own children as well. Approximately 70 per cent of all the cases of active reinfection pulmonary tuberculosis found in young people during the period of study in Washington County occurred among the children of men who were ill with or died of tuberculosilicosis (Table 7).

Table 6. *Results of Direct-Smear Examinations of Sputum of Patients with Tuberculosilicosis Dead Within a Two-Year Period of Observation.*

Age	No. of Known Dead	No. Permitting Sputum Examination	Cases with Positive Sputums
yr.			%
20 – 30	0	0	0
30 – 40	5	4	100
40 – 50	10	6	83
50 – 60	14	11	91
60 and over	12	9	78
Totals	41	30	
Average			87

The treatment of tuberculosilicosis is the same as that of pulmonary tuberculosis. The case with the early lesion of infection requires absolute bed rest to determine the stability of the lesion by daily temperature-recording, weekly weighings, sputum examinations and blood studies, and serial physical and x-ray examinations. A period of three to six months is often necessary to determine the character of the lesion. Collapse therapy can often be usefully applied in these early cases.

The more advanced cases are best handled by ambulatory methods whenever possible. But the common basis of treatment in these cases is properly applied and sufficiently prolonged rest. The duration of the bed rest varies with the patient, and cannot be accurately predicted because of the psychological problems encountered. Many moderately chronic cases show a definite decrease in dyspnea and cough following one month of rest in bed. Often, with the return of the temperature to normal and the disappearance of toxic symptoms, the patient can get up and start on graduated exercise. Many can be taught the necessary regularity of rest and work. Some of these patients after three months in the sanatorium are able to resume work. Following the recurrence of their previous debilitating symptoms, many return to the sanatorium. Again, after a period of one to three months of bed rest, the patient may go back to work. This procedure may last for several years, until continuous bed rest becomes necessary for the marked symptoms which no longer are relieved by any known form of therapy.

Table 7. *Study of Active Reinfection Pulmonary Tuberculosis in Children of Fathers Ill with or Dead of Tuberculosilicosis.*

Total cases of known active reinfection pulmonary tuberculosis in persons under 40 years of age in Washington County during period of study, not engaged in granite work	65
Number of these cases with father ill with or dead of tuberculosilicosis	45
Sex:	
Males	11
Females	34
Age group	
0 – 9 yr.	3
10 – 19 yr.	14
20 – 29 yr.	19
30 – 39 yr.	9

Summary

The pathogenesis and the occupational and environmental factors of tuberculosilicosis are discussed. The symptoms and signs and the roentgenological and laboratory findings are described, and the clinical focus defined, this being followed by a consideration of prognosis, prophylaxis and treatment.

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HYPOTENSION: THE IDEAL NORMAL BLOOD PRESSURE*

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CHICAGO

HYPOTENSION has been looked on as a disease entity ever since the days of Norris,[1] who described the "low-blood-pressure" person as one who lacked stamina, tired easily, complained of cold extremities and showed an inability to do prolonged mental or physical work. "They are not exactly ill; yet they are rarely well." This clinical picture of the hypotensive subject has been accepted quite generally,[2-6] and is the syndrome discussed in this paper. We are not concerned with such diseases as Addison's disease, coronary thrombosis, status lymphaticus, shock or infectious diseases which may be accompanied by low blood pressure. The problem we shall attempt to clarify is the state of the individual who has for a long time had a low blood pressure, and who may complain of lack of vitality and fatigue, vague digestive upsets and loss of weight.

ARBITRARY LEVELS OF HYPOTENSION

The dividing line between normal and hypotensive pressure has never been unanimously agreed on. The upper limit of systolic hypotension has been put as high as 120 mm. and as low as 90 mm. Some authorities, notably insurance actuaries, place the limit at a certain percentage below the average for the specific age. These limits are always arbitrary. No writer has ever given a scientific or objective reason for selecting one level in preference to another. Because the 110-mm. level is chosen by about half the observers, we have selected it as the point below which all pressures are tentatively called hypotensive. We do not imply that this level makes any significant division in the realm of blood pressure. It does not.

The level of diastolic pressure below which hypotension is said to exist is not so frequently suggested. Norris[1] places the limit as high as 80 mm., and Gelman[7] as low as 50 mm. However, because the 70-mm. diastolic level corresponds statistically to the 110-mm. systolic level, it has been tentatively selected for the purpose of this report as the upper level of diastolic hypotension.

INCIDENCE OF HYPOTENSION

Hypotension as just defined is not a rare occurrence. In a sample series of 10,883 persons,[8] representative of an urban group, the age-standardized incidence of systolic pressures under 110 mm. was 25 per cent, as shown in Table 1, and that of diastolic hypotension 34 per cent. Although the incidence of hypotension as reported by different authors varies from 2 to 55 per cent, there is general agreement[9-21] that the incidence is about 20 per cent.

TABLE 1. *Incidence of Hypotension among 10,883 Persons.*

BLOOD-PRESSURE LEVEL	CRUDE INCIDENCE	INCIDENCE STANDARDIZED TO U. S. POPULATION 1934, AGES 20–74	
	%	%	%
Under 110 mm. systolic			25.3
7478 men	21.8	20.5	
3405 women	38.8	36.0	
Under 70 mm. diastolic			34.1
7478 men	30.3	29.9	
3405 women	46.5	43.4	

The incidences here given are for all age groups. The well-known increased incidence of high pres-

*From the Department of Medicine, Northwestern University.
Assistance in the preparation of the charts and tables was furnished by the personnel of the Work Projects Administration (Project No. 465-54-3-37 [3]), under the sponsorship of the Institute for Juvenile Research, Dr. Paul L. Schroeder, director.

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sures in the older age groups would necessitate a decrease in the incidence of low pressures. In the series just mentioned (Table 2), the age-specific

TABLE 2. *Incidence of Hypotension at Specific Ages.*

Blood-Pressure Level	Ages in Years										
	20	25	30	35	40	45	50	55	60	65	70
	%	%	%	%	%	%	%	%	%	%	%
Under 110 mm. systolic											
7478 men	20	22	24	25	23	22	18	18	14	10	8
3405 women	56	49	44	42	33	25	25	16	11	11	25*
Under 70 mm. diastolic											
7478 men	43	37	33	29	25	25	22	20	28	29	17
3405 women	66	59	54	46	38	31	29	26	24	17	25*

*Insufficient distribution.

incidence of hypotensive persons decreased in the older ages, although the men showed no decrease of systolic hypotension until the age of fifty. The incidence of hypotensive women decreased more markedly, but it must be remembered that much of this decrease is due to the unusually high incidence of low pressures among young women. If the high pressures are excluded and only those individuals with pressures under 140 mm. systolic and 90 mm. diastolic are studied, the incidence of hypotension in the remaining more normal group illustrates even better the stability of low pressures throughout the life span (Fig. 1).

The occurrence of systolic pressures below 90 mm. is rare. Of 7478 men, only 42 (0.5 per cent) had such pressures. Of 3405 women, only 56 (1.7 per cent) had them. Only 2 of the men and 4 of the women had systolic pressures below 80 mm., and 16 men and 23 women had pressures between 80 and 85 mm. In the entire group only 30 men and 22 women had diastolic pressures below 50 mm. This does not mean that these persons always register such low readings. It is more probable that most of them, if studied over a period of years, would be found to be stabilized at the 100-mm. or even 110-mm. level, and these very low readings probably represent for the most part an exceptional downward dip.

The distribution of pressures under 90 mm. systolic, 50 mm. diastolic over the life span studied brings out an interesting comparison between men and women. Considering the age distribution of the total sample, wherein the bulk of the group was concentrated between thirty and fifty years of age, the men with systolic readings below 90 mm. showed no age differentiation (Tables 3 and 4). The frequency distribution of these readings follows that of the total sample. However, among women the low pressures were clustered in the earlier age groups, a distribution similar to that of the entire sample of hypotensive pressures.

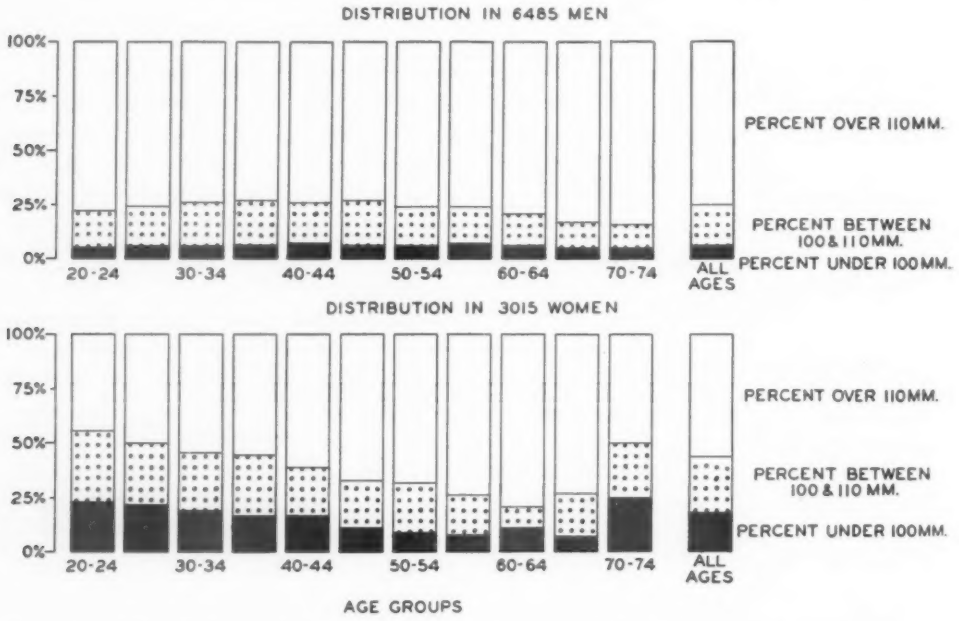

CHART 1. *The Incidence of Low Systolic Pressures in a Delimited Group of Adult Men and Women (pressures under 140 systolic, 90 diastolic).*

In men there is an almost even age distribution of low systolic pressures. Young women show much more low pressure than young men and old women show as much or slightly more low pressure than old men.

The Hypotensive Habitus

The person with low blood pressure tends strongly to be underweight.[22] Using the ponderal

index* as a measure of obesity, it is seen that while 30 to 60 per cent of underweight men had systolic pressures under 110 mm., only 10 to 17 per cent of the heavyweight men had them (Fig. 2). The underweight men showed from 40 to almost 80 per cent of low diastolic pressure, while the overweight men showed only 11 to 19 per cent.

Women with low blood pressure also tend to be underweight. Forty-four to 63 per cent of the lightweight women had pressures under 110 mm., while only 15 to 28 per cent of the heavyweight women had them. Similarly 55 to 69 per cent of the lightweight women had diastolic pressures under 70 mm., while only 23 to 35 per cent of the heavyweight women had them.

In respect to the build of the hypotensive person as differentiated from his weight, the chest-height index is used to delineate the linear or slender build (a low index) from the lateral or broad build (a high index).[23] From Figure 2 it is apparent that the hypotensive person falls in a linear-build classification twice as often as in a lateral-build one. Thirty-seven to 44 per cent of slender-build men had systolic hypotension, while only 14 to 26 per cent of broad-build men had systolic pressures under 110 mm. Also, 35 per cent of slender-build men had diastolic hypotension, while only 17 to 21 per cent of broad-build men had it.

In the group of slender-build women, 43 to 69 per cent had a systolic hypotension, while only 23 to 33 per cent of broad-build women had it. Also, 53 to 66 per cent of the former women had a diastolic hypotension, while only 29 to 39 per cent of the latter had it.

Extended Study of Low Pressures

The variation of blood pressure from day to day and from year to year in the same person is an outstanding characteristic of vascular tension. To a large extent the degree of variation, particularly of systolic pressure, is a characteristic of the level of pressure. An annual study of blood-pressure readings taken over a span of five to ten years revealed several significant facts. It was discovered that the year-to-year variation in blood pressure was much less among those persons who consistently registered low pressures than among those who consistently registered high pressures. Table 5 shows that greater yearly variations were found among the individuals with higher pressures. The

Table 3. *Distribution of Extremely Low Systolic Blood Pressures According to Age.*

Blood-Pressure Level	Ages in Years: under 20	20–24	25–29	30–34	35–39	40–44	45–49	50–54	55–59	60 and over	Totals
In 7478 men:											
Under 80 mm.	0	1	0	0	0	0	0	1	0	0	2
80–84 mm.	2	1	0	6	1	3	3	0	0	0	16
85–90 mm.	1	1	4	1	3	4	2	5	1	2	24
In 3405 women:											
Under 80 mm.	1	0	2	0	0	1	0	0	0	0	4
80–84 mm.	1	1	5	9	3	2	1	0	0	1	23
85–89 mm.	1	6	8	7	3	4	0	0	0	0	29

Table 4. *Distribution of Extremely Low Diastolic Blood Pressures.*

Blood-Pressure Level	Ages in Years: under 20	20–24	25–29	30–34	35–39	40–44	45–49	50–54	55–59	60 and over	Totals
In 7478 men:											
Under 50 mm.	5	7	4	6	2	2	2	1	0	1	30
50–54 mm.	8	15	21	18	17	10	11	5	4	7	116
In 3405 women:											
Under 50 mm.	2	2	6	6	2	3	0	0	1	0	22
50–54 mm.	14	28	44	39	18	22	8	3	2	2	180

Table 5. *Mean Yearly Variation of Systolic Blood Pressure at Various Levels.*

Blood-Pressure Level	No. of Men	Average Mean Yearly Variation
		mm.
140 mm. and over	28	13.0
130–140 mm.	40	9.9
120–130 mm.	78	9.1
110–120 mm.	116	9.8
100–110 mm.	90	8.0
Under 100 mm.	22	5.7

*Robinson, Brucer and Mass[22] have shown that weight alone is not an adequate measure of relative underweight or obesity. By dividing weight by height a more practical index of obesity can be obtained. An index number under 2.0 denotes a relatively lightweight person, while one higher than 2.4 denotes an obese person.

same is true for diastolic pressures, but to a lesser degree.

Furthermore, the average range of difference between the highest and lowest annual readings was less among those who registered low pressures than among those who had high pressures over a period of years but seldom experience a rise into hypertensive levels.

Thus it is apparent from this study of yearly variation that the dynamic behavior of low pressures is characteristically different from that of high pressures in every respect. Both low and high

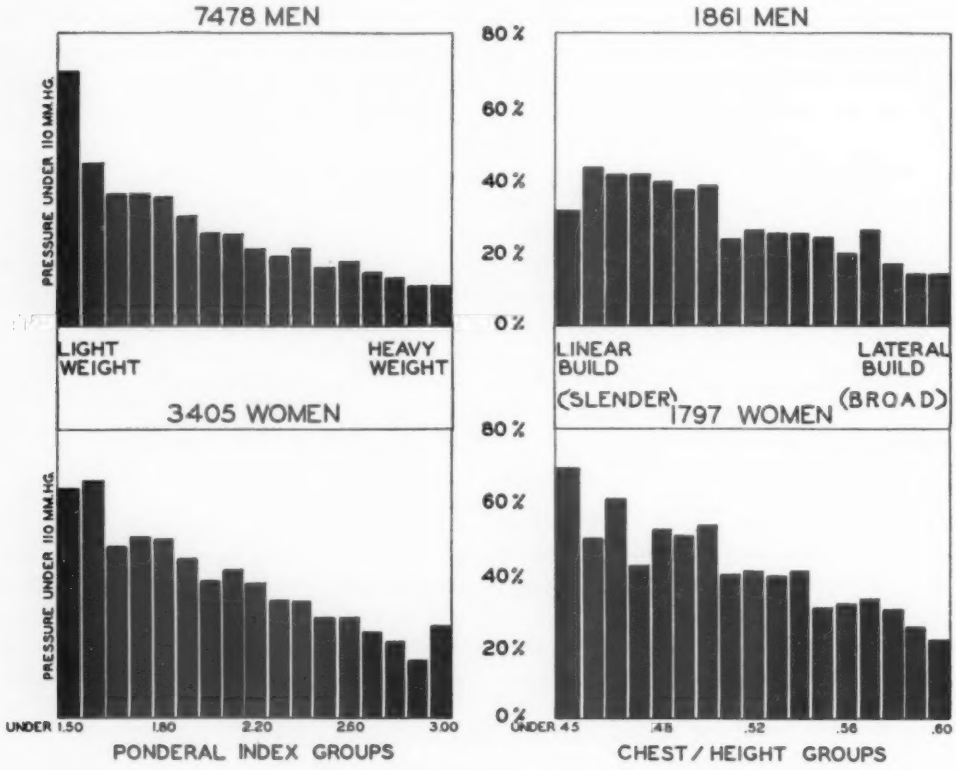

CHART 2. *The Incidence of Low Systolic Pressure in Various Weight and Build Groups. There are many more low blood-pressure readings among the lightweight than among the heavyweight men and women. There are also more low readings among the linear or slender-build than among the lateral or broad-build men and women.*

(Table 6). The diastolic pressure showed a smaller variation between the high and low readings. High diastolic pressures showed an average range of 15.9 mm., while low diastolic pressures showed an average range of only 12.7 mm. Finally, it was found that persons with the so-called "hypotensive" pressures not only tend to keep these pressures vary from year to year, but high pressures vary to a greater extent than do low pressures, and the variation is more erratic. High pressures show a tendency to ascend to higher levels over a ten-year span, while low pressures tend to maintain their level. Only rarely does a pressure that dips below 110 mm. ascend later to hypertensive levels, or does a pressure that is persistently above 120 mm. fail to ascend and become hypertensive (Table 7). Emotion is known to raise blood pressure, but the interesting fact is that persons with low pressures are usually affected very little by these excitations, and frequently not at all.

TABLE 6. *Extremes in Range of Systolic Blood Pressure at Various Levels.*

Blood-Pressure Level	No. of Men	Average Range of Difference between Lowest and Highest Readings over a 5-to-10-Year Span
		mm.
140 mm. and over	28	26.6
130–140 mm.	40	19.0
120–130 mm.	78	19.7
110–120 mm.	116	19.8
Under 110 mm.	112	15.3

Mortality Associated with Low Blood Pressure

The physician should not use the words "normal" or "abnormal" in connection with any physiologic level until he has investigated its associated

mortality. If low blood pressure is abnormal it should be associated with a high mortality. However, most medico-actuarial experience in blood-pressure levels speaks in favor of low pressure, as is shown in Table 8. In a recent study by Hunter,[24] the actual to expected mortality of systolic pressures 13 to 22 mm. below average was 81 per cent, and pressures from 3 to 12 mm. below average showed a mortality of 92 per cent, whereas the average experience was 102 per cent. The mortality studies of the insurance companies show that persons with low pressures have a lower mortality rate than have those with average or normal pressures. If the low pressures of the hypotensive group are associated with a more favorable mortality than is any other level of pressure, it would seem, at least from the standpoint of the end result, that the low pressures are the truly ideal ones.

TABLE 7. *Illustrative Records of Low Blood Pressures in Healthy Individuals.*

Case No.	Age at First Examination	Year													
		1925	1926	1927	1928	1929	1930	1931	1932	1933	1934	1935	1936	1937	1938
1	48	100/70	108/72	108/72	108/70	100/70	108/78	102/70	100/60	100/68	96/70	100/80	108/68	106/74	108/70
2	28			102/62	108/70	106/70	106/68	106/60	120/72	110/70	120/68	110/70	108/72	98/70	
3	32		112/84	114/70	100/68	100/70	98/70	102/78	98/70	92/68	100/74	104/74	108/68	92/68	
4	47			100/66	100/64	106/68	100/70	102/72	104/72	90/58	98/66	106/70	100/60	—	84/58
5	45				100/76	—	100/68	96/60	110/70	94/60	90/58	90/62	88/60	—	98/60
6	40			112/72	—	106/76	102/70	96/69	104/74	106/66	110/60	106/66	106/70	—	110/60
7	39				106/70	98/72	98/72	96/62	100/66	106/72	108/72	98/64	94/70	—	94/70
8	47					106/72	102/62	106/60	102/64	94/58	98/66	108/70	—	—	98/60
9	39					96/58	102/62	102/64	110/70	102/70	100/66	108/68	94/68	102/60	
10	39				106/70	98/72	98/72	96/62	100/66	106/72	108/72	98/64	94/70	—	82/54
11	27				110/60	96/66	90/60	100/70	96/64	80/54	96/66	106/70			
12	42					94/64	94/58	90/56	98/68	100/66	—	102/64	—	98/60	
13	34				86/60	96/52	100/58	96/50	86/50	82/48					
14	48					98/60	96/60	96/62	84/54	—	104/62	—	102/58	—	96/60
15	61				82/54	—	84/58	—	80/56	88/52	88/46	88/60	—	78/42	82/54
16	32				90/54	—	78/46	94/60	—	86/58	84/52	—	80/50	—	86/58

TABLE 8. *Mortality of Persons with Low Blood-Pressure Levels According to Various Insurance Companies.*

Company	Blood-Pressure Level	Mortality	
		Low pressure	Average pressure
		%	%
Northwestern Mutual[27]	100 mm. and under (systolic)	33	80
American Actuarial Society[35]	105 mm. and under (systolic)	47	86
	106 and 110 mm. (systolic)	65	86
Mutual Life of Canada[28]	10 mm. below average (systolic)	83	100
	15 mm. below average (systolic)	87	100
Joint Committee on Mortality[29]	5 to 15% below average (systolic)	94	100
Prudential Life[35]	Under 70 mm. (diastolic)	49	72
New York Life[30]	16 to 24% below average (mean pressure)	93	97
	5 to 15% below average (mean pressure)	91	97

The Clinical Picture of the Hypotensive Person

It is important to remember before condemning low blood pressure that the symptom complex attributed to the hypotensive person was arrived at through a general impression rather than through indisputable clinical statistics. These impressions are easily conditioned by our textbook heritage. For example, consider the common correlation of slender build, low pressure and fatigue. Of all slender-build, lightweight men, 44 per cent have a low pressure, and of all broad-chested, heavyweight men only 11 per cent have low pressure. Low pressure and slender build are clearly associated.[31] If a physician, trying to associate fatigue with low pressure and build, selected 1000 persons with low pressure for observation, 240 of these persons would be slender while only 60 would be heavy. If in each build group there were the same percentage of "fatigue cases," for example 25 per cent, 60 persons would show fatigue associated with low pressure and slender build, and only 15 with fatigue associated with low pressure and heavy build. Actually a conclusion from these data would be fallacious, since both the slender and the heavy groups had an equal percentage of fatigued subjects. It would not be fatigue that the physician studied, but rather the build-pressure relation. Thus it is seen how the clinical impression of a low pressure, lightweight and fatigue syndrome might develop.

The commonly accepted low-pressure symptoms, such as fatigue, headache and dizziness, are widespread complaints; there is no reason to ascribe them to any specific blood-pressure level. In fact, they are commoner in hypertensive than in hypotensive patients. A study of fatigue in 100 office patients showed that 22 per cent of the patients had low pressure, 49 per cent had moderate pressure and 29 per cent were hypertensive. Fahr[32] specifically states that it is usually the

hypertensive patient who notices tiring first. Davis[33] found "headache" to be present in 72 per cent of hypertensive patients. Riseman and Weiss[34] found headache and dizziness a part of the hypertensive picture in over 40 per cent of cases. Marshall[35] found that 62 per cent of hypertensive patients complained of headache; "general weakness" was present in 19 per cent. Ayman,[36] Gager[37] and Christian[38] all list the symptoms of hypertension as others do for hypotension. This is especially emphasized by the statement of Kylin[39]: "We find, therefore, that the hypertonic and hypotonic symptom complexes are similar in all essentials, except that the blood pressure is abnormally high in the one complex and abnormally low in the other."

We recognize that there are individuals who live in a hypofunctioning state. They may have a low blood pressure. They are nearly always underweight, and have a fragile appearance. They may complain of dyspepsia and nervousness. Their posture is frequently poor, and many are visceroptotic, have a low muscle tone and show evidence of a hypofunctioning glandular system. But these cases are rare and do not represent the typical hypotensive patient. Even in the rare cases of generalized hypofunction the low blood pressure is not the cause of the condition, but is only an incidental finding in a faulty neuroendocrine and biochemical physiology. Is it possible that the classic picture of the hypotensive person was derived from a study of this rare hypofunctioning type? It is idle, even in this type of patient, to institute any form of therapy directed at raising the blood pressure. Rather, the general condition should be improved by the use of insulin, high vitamins, adequate diet, ultraviolet light, and above all, graduated exercise.

Can the hypotensive person be an active individual, enjoy himself and undergo the rugged issues of life? This question can best be answered by citing some examples.

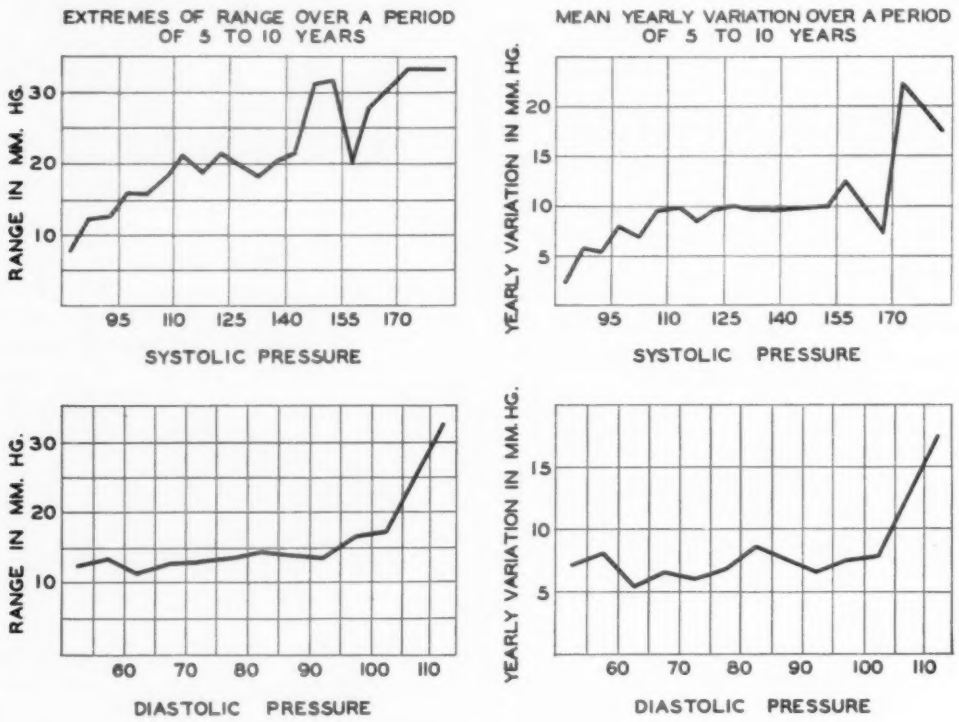

CHART 3. *The Variations of Blood Pressure as the Mean Level Rises. High pressures show less stability than do low pressures.*

CASE 1. O. K. M., a 32-year-old man, is 69 inches tall and weighs 150 pounds. He is a teacher of physical education and a coach of athletics; he conducts three daily calisthenic classes, demonstrates with his class on parallel bars, tumbling and horse work, and plays baseball and tennis. His blood pressure on two successive occasions was 98/58 and 98/56. He feels well and has no complaints.

CASE 2. R. J., a 46-year-old man, is 66 inches tall and weighs 155 pounds. He works at the International Harvester Company lifting 85- to 100-pound axles and steel frames into oil for tempering. He does his work fairly continuously all day. His pulse was 84 and his blood pressure 92/60. He feels well and has no complaints.

CASE 3. J. C., a 20-year-old boy, is 69 inches tall and

weighs 195 pounds, but does not appear to be overweight. He is extremely muscular and is active in athletics, baseball and track-team work. He does physical work but experiences no fatigue, and feels strong and healthy. His blood pressure was 88/58.

Case 4. A. I. N., a 44-year-old man, is 64 inches tall and weighs 145 pounds. He does exceptionally strenuous work carrying loaded milk boxes. His blood pressure on two consecutive occasions was 80/58 and 90/52. He stated that his blood pressure was low 10 years ago.

Case 5. H. F. J., a 26-year-old man, is 72 inches tall and weighs 172 pounds. His chest circumference is 40 inches and that of his abdomen 32 inches. A ballroom dancer, he does exhausting dance steps, with lifts, spins and sudden stops, and is especially strenuous in movements in which he throws and lifts his partner, who weighs 128 pounds. His blood pressure was 94/58.

Gregory[19] quotes the interesting case of a married woman who consulted him for the first time for her first pregnancy when she was twenty-five years old. She then had a blood pressure of 60 systolic, 45 diastolic. After attending her through three confinements and making numerous blood-pressure determinations, Gregory found that at no time except after exercise and during labor, did her pressure rise above 70 systolic, 55 diastolic. The interesting fact is that Gregory describes her as a well-toned, active woman who rode every morning and played tennis almost daily. Many similar records can be cited. Further evidence as to the desirability of low blood pressure is found in studies of physically active primitive people.

Nye,[40] studying physically active Australian aboriginals, makes the following observation: "In a general way the constant finding of a low-tension pulse and of arteries of normal appearance was striking. . . . Raised blood pressure and vascular sclerosis were entirely absent in this group of aged primitive aborigines." From this evidence he suggests that hyperpiesia is not a normal condition of senescence. He cites Rowan who found among these same aborigines only 1 case of sudden death and only 1 case of paralysis, in an old man. Donnison[41] found low blood pressure at all ages among active East African natives. Hudson and Young[42] found among the Arabs that 72 per cent had blood pressures of 110 mm. or under, and noticed relatively few diseases of the circulatory system. Ashford[43] found that the average systolic pressure of Puerto Ricans was well below the standards set in the United States. Fleming[44] made a study of Zuni Indians of New Mexico and found that in general their blood pressures were much lower than those encountered among white people. He was surprised to find many men

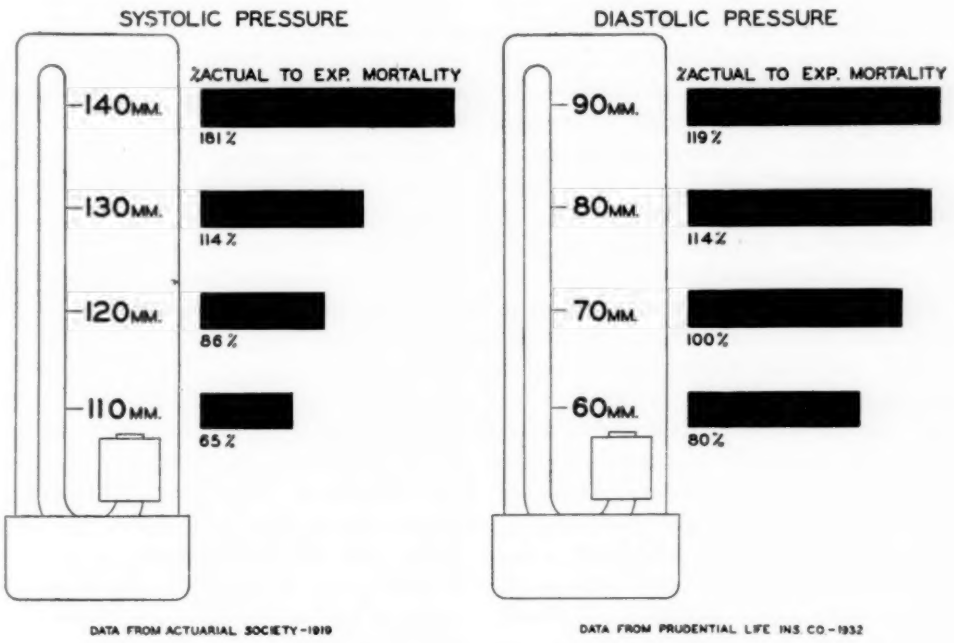

Chart 4. *Mortality Trends in Relation to Blood-Pressure Readings. This shows, for both systolic and diastolic pressures, that an increase in pressure is accompanied by a corresponding rise in death rate. The low readings are the most desirable as they are associated with the lowest mortality. (See Robinson and Brucer.[8])*

of approximately seventy years of age leading lives of extreme physical activity, performing feats requiring great endurance and registering systolic blood pressure between 110 and 125 mm. It should be apparent from this evidence that the classic description of the hypotensive individual has no evidence to support it.

Relation of Level of Blood Pressure to Vitality

In the past it has been widely believed that there is a causal relation between low blood pressure and fatigue, and it was consequently assumed that raising of the blood pressure would more adequately nourish the vital organs for more efficient work and greater vitality. As Bock[45] has stated, "Prior to the work of Dale and Richard and of Krogh it was assumed that the pressure of blood in the arterial system constituted the principal mechanism for the provision of an adequate supply of blood to the active tissues." Clinically this pseudophysiological concept has been carried over to support the contention that the hypertensive is active and energetic because of the high level of his blood pressure, while the hypotensive person is weak, ailing and lacking in stamina because he has not sufficient force behind his blood stream to circulate blood quickly enough for thorough oxidation. Johnson[20] has put it thus: "Hypotension takes so much of the joy out of living that its victim would gladly exchange [it] for the dynamic energy of his neighbor with a higher pressure."

However, it is to be questioned whether either fatigue or the sustained capacity for mental or physical work bears any true relation to the level of blood pressure. This problem has been answered by studies in the physiology of exercise. It has been shown conclusively that sufficient daily physical work raises the efficiency of practically all subjects without essentially altering their blood-pressure levels. This efficiency applies to cerebration and to the subjective state of the individual, as well as to his capacity for physical work. Bainbridge,[46] one of the outstanding authorities in this field, has stated, "Exercise not only leads to a greater sense of well-being, but enlarges the capacity for physical effort and without doubt results in optimum conditions under which the basic processes of life are carried on." Beyer[47] has said, "We sleep better, think better; we react, discriminate and associate ideas quicker; we see, hear and taste more distinctly . . . as an immediate consequence of exercise."

What are the changes in human physiology that occur when a sedentary subject with a low capacity for work or decreased physical fitness — for example, those with a low Schneider rating — is transformed by means of daily exercise or work to a better degree of health? These changes may be summarized briefly as follows: the resting pulse becomes slower and there is less increase in pulse rate from the recumbent to the standing position; the pulse and blood pressure increase less with exercise and return to normal more quickly after exercise; the stroke volume of the heart of the active individual is increased over that of the sedentary subject and he requires less oxygen. There is increased lung capacity, increased ability of muscle cells for oxidation and an increase in the capillary bed.[48–54] All these changes are accomplished, according to most observers,[45, 46, 55] without raising the resting blood pressure. In fact, some observers[56–59] have found that the blood pressure and pulse are lowered as physical fitness increases.

From the foregoing it is clear that low blood pressure does not cause fatigue and lessened vitality, any more than high blood pressure causes an increase in physical efficiency. The degree of health or mental and physical fitness is not an attribute of blood-pressure levels; rather it is dependent upon cellular oxidation and biochemical changes and these, in turn, aside from hereditary differences, are to a great degree dependent upon the extent of daily work an individual performs.

The physiological differences between the active and the sedentary person are of the greatest biological importance to man. They represent intrinsic and deep-seated advantages to the active person, all of which ultimately lead to a greater capillary bed and a greater utilization of oxygen by the tissues, measurable long after activity has stopped. These protoplasmic differences between the confined and active human being play a role in most of the degenerative diseases. Modern civilization, which imposes sedentary living on most city-dwellers, is the real cause for fatigue and lack of vitality, regardless of the level of blood pressure. Low blood pressure is found to be common in physically active persons such as farmers, primitive peoples and athletes; yet persons in these groups do not commonly complain of fatigue. It is easy to single out hypotension as the etiologic cause in any given patient's many complaints, because it is so easily and quickly measured. However, in most cases of fatigue and lowered vitality, regardless of the level of blood pressure, if we were to inquire further we should find a sedentary worker confined to a poorly ventilated office or shop, smoking a package of tobacco daily, riding to and from work, taking his recreation as a passive spectator rather than as an active participant and with practically no out-

door exercise. Fatigue and a condition below par more truly belong to such a combination of living habits, and are not to be attached specifically to one convenient cause, hypotension.

This may be well illustrated by the following thirty-five-year history of a Chicago attorney with so-called "hypotension." He is now fifty-four years of age, 69 inches tall, and weighs 149 pounds. His chest measurement is 34 inches, that of the abdomen 31 inches. He first learned of his low blood pressure in 1905 when as a student in college he was examined for participation in athletics. He was told that his systolic blood pressure was 104 mm. The examining physician, who was doing some research in blood pressure, checked him carefully for three months. His pressure was persistently low during this period, although he was allowed to participate in track and scrub football. He has been examined annually ever since, and his blood pressure has been constantly low. An examination each year since 1913 has shown that his systolic pressure has never exceeded 112 mm. Although he had never been sick in his life, he complained of fatigue and a "logy feeling" and lacked energy. He would fall asleep in the evening after dinner while reading the newspaper, and yet suffered from insomnia. In 1926 he bought a home with a large lot in the suburbs. He did all his own work, spaded, cut the grass, shoveled snow, bought a complete set of carpenter tools and did all the work around the house. He and his neighbor put up a steel-wire fence and sank the posts in concrete. He insulated his own garage and that of his neighbor. He does some physical work every weekday and all day Saturday and Sunday. Since he has become physically active he does not have any more fatigue. He can work through a twelve-hour day and still be on his toes all the time; his mind is clearer. With this subjective improvement of his health and greatly improved capacity for mental and physical work, his blood pressure remained unchanged. It was 98 mm. systolic, 68 mm. diastolic on September 20, 1939, and his pulse was 68.

This person was cured of his fatigue, low vitality and insomnia not by raising his blood pressure but by daily physical work. Just as low blood pressure cannot be blamed for the fatigue of the underweight person, high blood pressure (except extreme readings) cannot be held responsible for the fatigue of the obese person. Fatigue is not a function of the level of blood pressure; it is rather a deep-seated metabolic dysfunction most often related to inactivity.

Blood Pressure Below 90 mm. Systolic

Robinson and Brucer[60] have shown in a previous paper that blood pressures between 90 and 120 mm. were in the range of the normal. What about pressures below 90 mm.? Are they normal?

In the first place, we wish to distinguish two types of pressures at this level. There is the individual with a history of systolic pressure stabilized around 100 or even 110 mm. whose pressure in the course of a ten-year span occasionally dips below 90 mm. In many cases these occasional deviations can be accounted for. For example, the individual in Case 4 (Table 7) had a blood pressure stabilized over a twelve-year span at about 100 mm. systolic. In 1938 his level dropped to 84 mm., but this was a temporary fall following illness. We know that blood pressure falls during and following infectious diseases, especially influenza, and may stay low for a period of a few weeks or more. Hence, an occasional reading considerably below one's stabilized blood-pressure level may be caused by any acute illness, and is not a reflection of the intrinsic pressure of the individual. Such cases constitute the majority of systolic readings below 90 mm. The history of pressure just cited is definitely normal, as is shown from continuous studies of records and of mortality data.

Second, there is the individual whose history over a ten-year span shows a systolic blood pressure usually below 90 mm. Is this blood pressure normal? There is nothing in the literature to prove that it is abnormal, and all the phases of our study would indicate that it is normal. In the first place, there are very few such persons available for study. We have encountered only 4 out of the 500 persons in our continuous study covering five or more years. These individuals were enjoying good health and were actively at work. The literature contains other such records. If it is a fact that these persons are in good health and do not complain, and if their life expectancy fits in with what has been demonstrated for low blood pressures generally, there is no reason to look on these very low blood pressures as pathologic. Until evidence to the contrary is presented, we are practically compelled to accept this viewpoint.

Summary and Conclusions

Between one fourth and one third of the adult population have blood pressures under 110 mm. systolic and 70 mm. diastolic. Such blood pressures are usually designated as "hypotension."

Age has a negligible effect on the distribution

of low systolic blood pressure in men up to the seventh decade; thereafter the incidence decreases. Young women have a much higher incidence of low blood pressure than do men, and old women have about the same incidence as do old men.

Hypotension is commonest among the linear-built or narrow-chested type of build.

It is two or three times commoner among light-weight than among heavyweight men and women.

Low blood pressures show a smaller and less erratic yearly variation, and do not vary over as great a range as do high pressures.

They do not tend to rise with age; rather they maintain an even level.

They are associated with a mortality rate lower than that of average pressures.

The symptoms usually ascribed to hypotension are in reality commoner among hypertensive persons. There are no symptoms peculiar to or due to low blood pressure.

Neither fatigue nor vitality is a function of the level of blood pressure; rather they are related to the daily physical activity of the individual. In fact, those persons noted for endurance, vitality and well-being, trained athletes, farmers and other active groups, are also noted for their high incidence of low blood pressure.

Hypotension is not a disease; it is an ideal blood-pressure level.

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CLINICAL NOTE

A SYMPTOM OF ONE-SIDED AFFECTION OF THE LUNGS AND OF THE MEDIASTINUM

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IN MANY cases of chronic affections of the lung, pleura and mediastinum (basal pleuritic adhesions, chronic pneumonias, affections of the hilum lymph nodes, tumors of the bronchi and *Pleura-kappe*), I have very often witnessed a symptom which Pottenger[1] observed many years ago. Whereas in cerebral hemiplegias the protruded tongue deviates toward the opposite or paralyzed side, — for example, in the lesion of the left cerebral hemisphere the right half of the body is paralyzed, with deviation of the tip of the tongue to the right, — in affections of one side of the lung or of the mediastinum the tip of the tongue deviates to the affected side. The half of the tongue corresponding to the affected side is smaller than the other half. Sometimes there is a slight fibrillary twitching of the muscle fibers, and the border of the tongue may be shallower on the deviated side. No sensory defect can be detected in either the anterior or the posterior part of the tongue.

Since hemorrhage into the internal capsule involves supranuclear hypoglossus fibers situated close to the genu (geniculate fibers), there results a paralysis of the hypoglossus on the same side as the paralysis of the body muscles. This paralysis is characterized by deviation of the tip of the tongue to the paralyzed side, since the normal musculature of the opposite genioglossus muscle pulls the tip of the tongue forward and at the same time toward the paralyzed side.

Cortical lesions present the same symptoms so far as the paralysis of the musculature of the body of the tongue is concerned. In severe cases of cellulitis of the neck there may result a peripheral paralysis of the hypoglossus nerve, due to phlebitis or periphlebitis of the jugular and facial veins. In toxic neuritis — that caused by the ingestion of lead, arsenic or alcohol — a peripheral toxic paralysis of the hypoglossus nerve may result which escapes the observation of the patient because the intervening of the muscle fibers of both halves of the tongue prevents any functional disturbance.

For the interpretation of the above-mentioned sign, two factors may be considered. First, the underlying inflammatory or neoplastic condition of the lung or surrounding tissues may produce a hematogenous damage to the hypoglossus nerve on one side. (Chronic polyneuritis combined with muscular atrophy is not uncommon in chronic pulmonary tuberculosis.) The fibrillation of the muscle fibers of the tongue could be interpreted in this manner, since in toxic neuritis myokymia is not uncommon. As swelling of the lymph nodes in the mediastinum, in the neck and at the base of the skull in chronic pulmonary affections is not rare, a compression of the hypoglossus nerve by a lymph node could be possible.

Since the atrophy of one half of the tongue and the deviation of its tip correspond regularly to the position of the affected lung, a reflex may be significant. Afferent vagus fibers from the lung irritated by the chronic process within the thorax might transmit the irritation to the hypoglossus nerve, since anastomoses between the vagus and the hypoglossus nerves, after the latter has left the hypoglossal canal, and between the hypoglossus nerve and the ganglion nodosum are common.

The syndrome of deviation and atrophy of one side of the tongue seems to belong to the old, vegetative, accompanying symptoms of chronic lung diseases, such as Horner's syndrome, atrophy of the trapezius muscle of one side, redness of the cheek, difference between pupils and so forth. The syndrome is of diagnostic value.

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REPORT ON MEDICAL PROGRESS

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Tetanus Toxoid

ACTIVE immunization against tetanus among selected groups has been employed for several years in certain European countries. Those receiving this treatment have been persons whose duties necessitated frequent exposure to infection. The results of this type of prophylaxis have been so satisfactory that several observers in this country have been stimulated to make further observations.

Active immunization may be obtained by the subcutaneous or intramuscular injection of tetanus toxoid, plain or alum-precipitated. The latter is more slowly absorbed and hence exerts an antigenic stimulus over a longer period of time. For this reason it has been considered by most observers to give a higher antitoxic response than does plain toxoid.

The studies of Gold,[1] Bergey and Etris,[2] Hayden and Hall[3] and others have shown that the highest antitoxic response is obtained when the injections are given at intervals of two or three months rather than two or three weeks. In fact, Jones and Moss[4] have shown that even a smaller total quantity of tetanus toxoid given at wide intervals produces a higher and more prolonged antibody response than does a larger volume given at a single injection. These observations are similar to those noted by Benjamin, Fleming and Ross[5] in a study of 5195 children who had received injections of diphtheria toxoid. It would therefore appear that the proper procedure is to give an initial intramuscular injection of 1 cc. of alum-precipitated toxoid and to repeat this dose either two or three months later.

There is very slight antibody response to the initial injection of toxoid, and it is only after the second injection that a satisfactory response may develop. In a study of 34 adults, Gold[6] showed that the tetanus antitoxin content of the blood fell rapidly during the first three months following the second inoculation, reaching less than 0.01 units per cubic centimeter of blood serum. In a subsequent study the same author[7] concluded that the minimal antitoxic value for protection should not be below 0.1 unit per cubic centimeter of blood serum. These observations are in accord with those of Hayden and Hall, who immunized 152 volunteers from the United States Military Academy. Six weeks after the first injection the tetanus antitoxin content of the blood rose from an untitratable quantity to one of about 0.01 unit per cubic centimeter, and eight weeks after the second injection to 0.40 unit. All observers agree that the content falls rapidly during the first few months following the last injection. However, an extraordinarily rapid increase in tetanus antitoxin content occurs following a third or stimulating dose, the level reaching as high as 10 or more units per cubic centimeter. This is more than would have been present following the administration of a prophylactic dose of antitoxin, and its presence is therefore of therapeutic value.

This antitoxic response develops in six to eight days following the injection of the third or so-called "stimulating" dose, and hence falls within the usual incubation period of tetanus. Such results, however, should not be expected when the inoculation is given sooner than one month after the second prophylactic injection. Also, as pointed out by Gold, this procedure is of no value if employed between the first and second immunizing vaccinations.

Recently a biological product has been made available which contains both diphtheria toxoid and tetanus toxoid. Hence the possibility of immunizing individuals, especially children, to both diseases presents itself. The question of the advisability of the administration of more than one antigen at the same time arises; in an effort to answer it, a large amount of experimental data has been accumulated. These data indicate that the antibody response to each antigen when two or more are employed is as high, and as prolonged, as when only one is injected. Jones and Moss[8] studied the antitoxic titers of 41 students at the Indiana University School of Medicine who received combined diphtheria and tetanus toxoid, and obtained results indicating not only that the combined use of the two toxoids was feasible, but that immunity to both diseases could thus be established. In a study of the same group one year later, it was shown that immunity to diph-

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theria had been satisfactorily established and that the response to the third or stimulating injection of alum-precipitated tetanus toxoid increased "the antitoxic content of the blood twenty to fifty times, making a level which is higher and more persistent than that produced by a prophylactic injection of tetanus antitoxin." From these studies it would appear that the combined use of the two toxoids produces a satisfactory immunologic response and is therefore of prophylactic value.

The universal administration of alum-precipitated tetanus toxoid, either alone or in combination with alum-precipitated diphtheria toxoid, may not be acceptable to all physicians concerned with the care of infants and children. In fact, there may even be certain objections to such a procedure. However, the administration of tetanus toxoid to all infants and children who are known to be allergic or who have previously received a dose of horse serum and are therefore probably allergic to it, or who are repeatedly being exposed to possible infection, appears to be advisable. Neither anaphylaxis nor serum sickness is agreeable. One should realize that there is a definite individual variation in the degree of antitoxic response to the injection of tetanus toxoid and that as yet there is no clinical test like the Schick test for diphtheria, by means of which this response may be determined; titration of the blood serum is the only means. Therefore, if a patient who has previously received two injections of toxoid is wounded under circumstances indicating that tetanus spores are likely to be present in the wound, it is better to give not only 1 cc. of tetanus toxoid but also a prophylactic dose of tetanus antitoxin.

Acute Anterior Poliomyelitis

It is an irrefutable fact that the diseases which are accompanied by disabling sequelae attract the greatest interest. To date one may truthfully say that infantile paralysis continues to occupy a prominent position among such conditions. Furthermore, it is unfortunately true that our knowledge concerning any specific form of therapy to be applied during the acute stage of this disease is embarrassingly scanty. However, an understanding of the epidemiology of the disorder, and particularly of the mode of dissemination with possible subsequent infection, may easily be a more effective measure in the control of acute anterior poliomyelitis than are drugs. Certainly this seems at present to be the most promising approach.

For many years it has been the common belief that the virus of acute anterior poliomyelitis enters and leaves the body through the respiratory passages. Recently attention has once more been focused on the possible presence of the infecting agent in the stools of patients with the disease, of convalescent patients and of persons without the disease who have been in contact with patients so infected. Harmon[9] in 1937 and Trask and his co-workers[10] in 1938 successfully demonstrated the presence of the virus, thus reaffirming earlier observations. In a later report the latter authors[11] studied thirteen stool specimens obtained from 8 patients and were able to isolate the infective agent in three specimens, all obtained from the same patient. It is significant that this patient did not have paralytic poliomyelitis (as his brother had at the time) but was suffering from the abortive form of the disease. As the authors point out, such a diagnosis might not have been made had not the virus been found. It was present in the stools as late as the twenty-fourth day after onset of the illness, thus demonstrating how long the patient might have served as a source of infection to others. A specimen of the stool preserved in a refrigerator for ten weeks showed the virus to be still viable. Successful recovery of the virus from feces has been reported also by Kramer,[12] Toomey[13] and Lepine.[14]

Kramer, Gilliam and Molner,[15] while studying an outbreak of the disease in a children's home in Detroit during the summer of 1939, were able to demonstrate the presence of the virus in the stools of 6 out of 19 persons who were not suffering from the infection but had been in contact with 5 patients who were. In one of these healthy persons the virus persisted in the stools for nineteen days following its first detection, and in another for thirty-eight days. Although it cannot be proved that these 6 people acquired the virus from the ill patients, it is reasonable to assume so. Thus it would appear that during an epidemic the patient is not the only source from which infection can be disseminated.

These recent observations may be of real epidemiologic significance, and naturally open further fields for study. From the practical aspect, consideration should be directed toward the proper disposal of the intestinal contents of all patients suffering from acute anterior poliomyelitis, a precaution which probably has not been considered very seriously in the past. Similar disposal of the feces of all healthy persons in contact with an ill patient would hardly seem practicable, and certainly is not so where large groups or institutions are concerned.

Since the virus may be present in the gastrointestinal tract of those who present no evidence of infection, as well as of those ill with the disease, the question of sewage as a possible mode of dissemination arises. Paul, Trask and Culotta[16] studied specimens of sewage obtained from dif-

ferent locations in the city of Charleston, South Carolina, during the summer epidemic in 1939 and were able to isolate the virus. In fact, their results demonstrated massive contamination of the sewage. Such observations naturally attract the attention of public-health officials as indicating a potential danger; indeed, the condition may exist in small localities or in situations where adequate sewage disposal is difficult. However, in most instances the sewage would be diluted to such an extent that the possibility of infection from this source would be reduced to a minimum. Naturally one would not advocate bathing in a small stream or body of water into which the city's sewage emptied. In view of our present knowledge, such procedures should be prevented during an epidemic. When one considers the large number of people attending the public beaches during the summer months, one cannot help but wonder if the chance of infection from spray-borne material from unrecognized carriers is not greater than that of infection from pollution of the water.

Pneumococcal Pneumonia

In the 1939 progress report,[17] attention was directed to the use of a new agent, sulfapyridine, in the treatment of pneumococcal pneumonia. Since that time another medium of chemotherapy has been offered which appears to possess many features that render it preferable to sulfapyridine. This new drug, sulfathiazole, is now commercially available. Its addition to our armamentarium gives us three forms of specific therapy for the treatment of pneumococcal pneumonia; antipneumococcus serum, sulfapyridine and sulfathiazole.

When so many promising procedures are available for the treatment of a disease, some confusion may result as to which is the best one in a given case. Blake[18] has recently analyzed the results obtained from the three drugs mentioned, and has pointed out the varying factors observed in the clinical response to each. He studied 250 cases of pneumococcal pneumonia treated at the New Haven Hospital. One hundred and nine patients received sulfapyridine alone, 41 sulfapyridine and antipneumococcus serum, 97 sulfathiazole alone and 3 sulfathiazole and antipneumococcus serum.

The 150 patients receiving sulfapyridine alone included 45 on the Pediatric Service; the rest were over the age of thirteen, the age limit for this service. Age appeared to be one of the most important factors in determining the outcome. Ninety-five per cent of the children were under ten, and only 3 per cent of these showed relapses, complications or fatal terminations following therapy. Unfavorable reactions, such as persistent low-grade fever, complications and death, paralleled the increase in age.

The results from treatment with sulfapyridine and antipneumococcus serum (41 cases) showed little if any difference from those obtained from sulfapyridine alone, and whatever difference was noted was in favor of the latter. However, a study of the results in this group discloses two striking facts, namely, that there were few patients under ten years of age, and that many cases suffered from an accompanying bacteremia. These are indeed important factors for a final appraisal of the statistics.

In the group of 100 patients who received sulfathiazole, the results were similar to those obtained with sulfapyridine alone, the difference again being dependent on age. As in the other groups, older patients who were suffering from Type 3 pneumococcal pneumonia showed a tendency to recover less rapidly than did those suffering from other types. A final study of the factors influencing mortality revealed that old age, with its accompanying incidence of chronic disease, was more important than bacteremia, the type of pneumococcus or the time, that is, day, relation of the institution of specific therapy to the duration of the illness.

The value of anti-pneumococcus serum in the treatment of pneumococcal pneumonia is beyond question. However, in dealing with specific forms of treatment in this disease, one must bear in mind that the prognosis of acute primary or lobar pneumonia in infancy and childhood differs from that among adults. Prior to the introduction of specific serum the mortality rate of this type of pneumonia among infants under two years of age was discouragingly high. Curnen[19] reports that in a series of 50 infants suffering from Type 14 pneumococcal pneumonia who received serum there were no deaths, a fact which he took to prove the value of this form of therapy. Yet no one who has administered serum to infants is unaware of the reactions, not including serum sickness, which often appear. Davies[20] observed equally good results when sulfathiazole alone was used in the treatment of 150 infants and children suffering from pneumococcal pneumonia. In this group, practically all recognized types of pneumococcus were encountered. This is a significant feature, for not infrequently many types of pneumococcus are present at the same time, a finding which makes it difficult to decide which type of serum to administer. Among infants with pneumonia, Type 14 is most commonly encountered, whereas in children over two years of age, Type 1 is usually the predominating organism. However, with rising age there is an increase in the number of types of organism present, and this of course

renders specific therapy with serum somewhat confusing. Fortunately, the use of sulfapyridine and sulfathiazole has obviated this difficulty. Davies's study showed that the temperature returned to normal fairly promptly, and that the majority of the patients were restored from a critically ill state to one of comparative comfort within twenty-four to forty-eight hours after treatment was instituted. This should not be misconstrued as necessarily implying that the clinical or roentgenologic evidence of pulmonary involvement disappeared in as short a period of time.

The dosage as well as the manner of administration of sulfathiazole is similar to that employed with sulfapyridine, that is, 1½ gr. per pound of body weight as the initial dose, followed by 1 gr. per pound per day, usually divided into six doses and administered every four hours, as the daily maintenance dose. In cases in which nausea is present it is advisable to divide the initial medication into two parts, giving half the total amount each hour for two doses. This produces a blood level between 4 and 11 mg. per 100 cc., which in most cases proves therapeutically effective.

Untoward, toxic manifestations, such as nausea, vomiting, fever, eruptions, hematuria and neutropenia, have been much less frequently encountered with sulfathiazole than with sulfapyridine. This fact, together with the clinical evidence that sulfathiazole is equally as effective as sulfapyridine, has made treatment with the former the procedure of choice for pneumococcal pneumonia occurring among infants and children.

Influenzal Meningitis

Although the administration of anti-influenza horse serum has to some extent reduced the mortality rate of influenzal meningitis, the results are far from satisfactory. The introduction of chemotherapy naturally prompted some investigators to try this type of treatment; although an occasional recovery has been reported,[21] failures are not unknown.[22] At the present time there is insufficient data even to suggest that this form of therapy will prove effective. The most promising type of treatment has recently been offered by Alexander and Heidelberger,[23, 24] who have prepared a successful anti-influenzal serum from rabbits. This possesses a smaller anti-carbohydrate molecule than does anti-influenza serum prepared from horses, and hence has greater power of penetrating the tissues. The specificity of this serum for Type B strains of *Hemophilus influenzae* can be demonstrated by microscopic studies executed in a manner similar to the typing of pneumococci by the Neufeld method. Dr. Alexander kindly furnished this serum for the treatment of 3 patients suffering from influenzal meningitis at the Infants' and Children's Hospitals,* and 2 of these patients recovered. The third child was moribund at the time of admission and died within a few hours.

It is well recognized that the mortality rate of this disease is influenced not only by age but also by season, so that care must be exercised in drawing conclusions too hastily. That such care is being exercised is clearly shown in the following statement by Alexander[25]: "While the number of patients treated is still very limited, the serum has been used with promising results in several clinics. When all types of cases are included, even those dying within less than twenty-four to forty-eight hours of admission, the resultant mortality has been approximately 50 per cent." Certainly such results justify further use of this type of anti-influenza serum.

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*This serum is now available commercially through E. R. Squibb & Sons, New York City.

CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTEMORTEM AND POSTMORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

CASE 26371

PRESENTATION OF CASE

An eighteen-year-old Jewess was admitted to the hospital complaining of intermittent joint pain.

The patient was well until nine months before admission when she awoke one morning to find "a funny aching pain" in both knee joints, made worse by exercise. So far as she could recall, the knees were not swollen, red or hot, and the pain was transient, having periods of exacerbation and remission. She went to bed for a week, rested two more, and then returned to school, entirely free of joint symptoms. She engaged in normal scholastic athletics, such as bowling and gymnasium exercises, without joint discomfort. She was free of symptoms for about two months; then, approximately seven months before admission, she again began to have pain in the knees, with occasional puffiness and tenderness to touch and movement, but with no noticeable redness of the overlying skin. She soon experienced similar discomfort in the wrists and proximal phalangeal articulations. The wrists caused so much pain for a short while that she taped them with adhesive in the belief that they were sprained. Small red spots appeared at the sides of several of the involved proximal joints of the fingers. Following this the ankles, elbows and wrists intermittently became tender and painful on motion. The attacks of pain were transient, lasted only a few days and tended to involve one joint after another. Although never considered a sickly girl, she had been extremely susceptible to upper-respiratory infections. She had had a tonsillectomy in infancy. There was no history of nosebleeds, rheumatic fever or chorea. Familial diseases included "arthritis" in a maternal uncle and grandfather, and hay fever in a maternal aunt. The remainder of the past and family histories was non-contributory.

Physical examination revealed a well-developed and well-nourished girl who lay quietly in bed in no discomfort. She had a rather dusky color; there was no pallor of the mucous membranes. The heart was not enlarged; the rhythm was regular, the rate being 142. The sounds were of fair quality. There was a faint, apical systolic murmur and a roughening of the pulmonic second sound, which was louder than the aortic second sound. The blood pressure was 126 systolic, 72 diastolic. Examination of the lungs was negative. The liver was palpated a handbreadth below the costal margin, and the tip of the spleen was felt. Pelvic examination was negative. There was a slight right upper and left lower dorsal scoliosis. Difficulty was experienced in fully extending the fingers of the right hand, but no limitation of motion of any other joints was noted but there was pain on moving the knees and elbows. Both feet and lower legs were colder than the remainder of the body. Epitrochlear lymph nodes were palpated, but no rheumatic nodules were noted.

The temperature was 101°F., the pulse 120, and the respirations 24.

Examination of the blood showed a red-cell count of 4,200,000 with 74 per cent hemoglobin, and a white-cell count of 8800 with 64 per cent polymorphonuclears, 15 per cent lymphocytes, 4 per cent monocytes and 17 per cent eosinophils. The platelets were normal. The urine examination showed a specific gravity of 1.038, with 10 to 20 white blood cells per high-power field in the sediment; there was no albuminuria. The stools were guaiac negative. A blood Hinton test and a gonococcal complement-fixation test were negative. Frequent blood cultures were negative. The blood sugar was 92 mg. per 100 cc., the serum protein 6.9 gm., the serum albumin 3.2 gm., and the serum globulin 3.6 gm. or an albumin-globulin ratio of 0.89. The serum nonprotein nitrogen was 17.2 mg. per 100 cc., the serum calcium 10.2 mg., the serum phosphorus 3.9 mg., and the uric acid 3.7 mg. A sedimentation rate in fifteen minutes was 20 mm.; thirty minutes, 49 mm.; forty-five minutes, 54 mm.; sixty minutes, 56 mm. An electrocardiogram showed a ventricular rate of 95 with normal rhythm, a slight tendency to left-axis deviation, a complete inversion of Lead 3, but an otherwise normal record.

Roentgenograms of the hands and knees showed slight soft-tissue thickening about the interphalangeal joints of both hands. The cartilage spaces, however, were of normal width. The articular surfaces were smooth, and the bones were not remarkable. The bones of both knee joints showed no definite variations from the normal. A chest plate showed that the heart shadow was at the upper limits of normal. The heart was broad across the base, and there was definite prominence in the region of the left ventricle. The lung fields were clear. The diaphragm was normal in position and outline.

The patient ran an almost constant, slightly elevated temperature (around 100°F.), with a

slightly elevated pulse (about 110) and respirations which varied from 22 to 25. After a few weeks in bed she began to ooze blood from the nose and mouth. It was then found that the blood clotting time was 40 minutes, and the bleeding time 13½ minutes; a tourniquet test was positive, with many petechiae. The platelets were normal in number, and the clot retraction was good at 4 and 12°C. The prothrombin value was 32 per cent of normal, and the antithrombin factors were greatly increased. The hematocrit was 34 per cent. The plasma vitamin C determination was 0.29 mg. per 100 cc., and the total carotinoids were 0.04 units per cubic centimeter (the normal being 2 to 4 units). Vitamin A determinations showed 0.6 units per cubic centimeter (the normal being 1 to 4 units). These findings indicated a deficiency in vitamins A, C and K. She was given large quantities of vitamin concentrates, with methyl naphtholquinone and dioxycholic acid by mouth. Later, methyl naphtholquinone was given parenterally, and she was transfused. With this treatment she ceased oozing blood, but three to four weeks later the symptoms recurred and required further vigorous though similar therapy.

The liver and spleen became progressively enlarged. A cervical lymph node removed for biopsy showed hyperplasia. An aspirated specimen of the sternal bone marrow was normal. A biopsied piece of muscle showed no diagnostic abnormality. Blood smears continued to exhibit a relatively high eosinophilia, 22 per cent on one occasion. A van den Bergh test showed 4.1 mg. bilirubin per 100 cc. There was a Congo red retention of 67 per cent, a bromsulfalein retention of 40 per cent, a +++ Takata-Ara test, biliuria, hematuria, a basal metabolic rate of +18 per cent, normal blood sugar, a normal hippuric acid excretion and a negative 17-ketosteroid determination. An undulant fever agglutination test and tests for kala-azar were negative.

A transient but definite pericardial effusion developed and slowly receded during the middle of her hospital stay. She developed a chronic mouth infection and seemed to be in extremis on two or three occasions, with high temperature, great prostration and mental disorientation. On each occasion she rallied and seemed to return almost to her former fair health. At one time she developed a rash on the hands, the left elbow and the buttocks. This rash consisted of irregular erythematous patches which were not infiltrated over the buttocks but which showed definite thickening and infiltration elsewhere. She slowly but gradually failed, became dyspneic and had irregular respirations. About two weeks before death she developed diarrhea. At that time proctoscopic examination showed a "dry" glazed mucosa with a tenacious adherent brownish discharge becoming more liquid as one proceeded upward to the level of the rectosigmoid junction. The swabs removed at proctoscopy were guaiac positive. No discrete ulcerations or polyps were observed. The abdomen became distended and tympanitic, with rare peristaltic sounds. The liver and spleen continued to become more palpable. Edema of both legs appeared. There were anorexia and frequent bowel movements, accompanied by occasional incontinence of foul-smelling fecal material. The blood smear showed immature white cells, and the eosinophilia disappeared. She gradually failed, and died about four months after admission.

Differential Diagnosis

Dr. William B. Breed: This patient was on the ward when I came on service and was there when I went off. Practically everybody on the medical services at one time or another wrote words of wisdom about her, and I hope I shall be permitted to read my own notes at this exercise. I have not learned what Dr. Mallory found; and so it was considered legitimate to modify the technic of these exercises a bit by allowing me to discuss a patient with whose clinical course I am familiar.

It appears from the record that this girl was sent into the hospital for study, probably with a question of rheumatic fever, rheumatoid arthritis or subacute bacterial endocarditis. I believe there was no definite diagnosis made before she was admitted. In the history of the present illness, rheumatic fever and rheumatoid arthritis are the two most likely diagnoses that are suggested. The physical examination brings up at once the question of subacute bacterial endocarditis. It is only when one learns that, after she had been in the hospital for some time, evidence accumulated that a number of systems had become involved that one goes on to a more complicated diagnosis.

It is obvious that the liver and spleen were involved, and there are symptoms and signs suggestive of endocarditis. Scoliosis is mentioned, but no abnormality in any of the joints. I can tell you that, directly she was admitted, she went slowly downhill and developed evidence of widespread systemic involvement, characterized by bleeding from the mucous membranes, considerable anemia, pericarditis with effusion, still further increase in the size of the liver and spleen and, at one time, a rash on the arms and buttocks. Of course, it seems impossible that this

whole picture could have been due to simple rheumatic fever or rheumatoid arthritis, and so with just a little reservation on rheumatic fever, —the malignant type,—I shall let both of these go.

In discussing the laboratory work you will notice that she had persistent eosinophilia until the very last, which suggests at once two important diseases, namely, trichinosis and periarteritis nodosa. There was no history pointing to trichinosis. A biopsied piece of muscle was examined and found to have no cysts in it; and trichinosis is not a disease which will produce this whole picture. So the explanation of the eosinophilia rests rather strongly with periarteritis nodosa. She then developed this rash, which we had looked for quite often in the hope that we might be able to establish a diagnosis of acute disseminated lupus erythematosus, a syndrome that was very high on the list of possible diagnoses. The kidneys were functioning well. She had an increased sedimentation rate. She had an inverted albumin-globulin ratio. There was no evidence of parathyroid disease by chemical tests. Toward the end she had prolonged clotting and bleeding times. It is interesting that with this increase in clotting time the clot retraction and platelets were said to be normal, a fact which I think rules out a true purpura reasonably well. It is obvious that she had severe avitaminosis; and that brings up the question which is becoming so prominent today: namely, How easily is the vitamin reserve depleted in a severe infection? I believe that the bleeding which occurred from the mouth and the accompanying local infection were caused by a depletion of vitamin C or K, or both, and not due to some fundamental blood dyscrasia. I just call attention to the statement "negative 17-ketosteroid determination"; I take it that means the steroid was present in the urine and that she did not have adrenal disease.

Dr. Sedgwick Mead: Dr. Parsons tells me that there was a definite reduction of 17-ketosteroid.

Dr. Breed: A "negative" 17-ketosteroid determination does not mean anything. The fact that it was reduced suggests that the function of the adrenal glands was decreased in the presence of this infection, and that she did not have primary adrenal disease. Of course, the function of all the organs was decreased, and it is perfectly reasonable to expect that the function of the adrenal glands would also be lowered. She developed diarrhea and was proctoscoped, and no doubt the question of chronic ulcerative colitis was brought up. There was a glazed mucous membrane and the swabs showed blood, but there again I think the colon took part in this generalized infection, and it is not legitimate to make a basic diagnosis of ulcerative colitis.

I should like someone to comment on the effect of the transfusions that this girl had; little appears in the record. At one time she was given fourteen transfusions in about as many days and showed a marked remission. The fact that she was returned to nearly normal health was due in our opinion to the repeated transfusions. After that she slumped again and I believe was given another series of transfusions.

Dr. Wyman Richardson: She was given many more.

Dr. Breed: Did she react as well to the second series as she did to the first?

Dr. Richardson: No, although there was temporary slight response.

Dr. Breed: It is clear that only two diagnoses occurred to me as being important. These were acute disseminated lupus erythematosus and periarteritis nodosa. The appearance of the rash, although not typically distributed, and its disappearance with transfusion convinced me, as I left the case, that she was suffering from the former. I shall read what I wrote in the record: "Persistent eosinophilia, large liver and spleen, hematuria and other bleeding in the past, taken together with a negative lymph-node biopsy and a negative sternal bone marrow, leave periarteritis nodosa almost alone as a possibility. Suggest muscle biopsy."

Fourteen days later I wrote: "We cannot eliminate either lupus or periarteritis nodosa. The rash is not characteristic of lupus but lends some support to the diagnosis. Let us remember that often the pathologist cannot distinguish between these two syndromes."

Dr. Tracy B. Mallory: I am going to challenge that statement, Dr. Breed.

Dr. Breed: Well, at least we have a challenge.

In casting about for other diagnoses, unusual to be sure, because this is an unusual case, I suppose one has to remember that she might have had tuberculosis. The fact that she responded rather remarkably to transfusion once and the fact that we never could make a diagnosis of tuberculosis while she was alive seem to make that quite unlikely. So I still am bound to rest on these two diagnoses, and I think now that if I had to put one before the other I should put lupus first and mention periarteritis nodosa, but not with any enthusiasm. I have no criticism to make of the treatment. I think she had about thirty transfusions—the only lead we had in the way of specific treatment in the hope that she might stage a more prolonged remission.

DR. MALLORY: As Dr. Breed has said, this patient was in the hospital several months and nearly everyone on the service saw her; a large proportion of them committed themselves to a note in the record. I might ask Dr. Richardson to express an opinion.

DR. RICHARDSON: I was on service when this patient died and I also made various diagnoses. She had during the last month of life an abnormal blood picture in that there were present quite numerous, very immature cells, some of them of the blast type. The blood picture, however, was quite suggestive of infectious mononucleosis. She did have a sore throat, and we went so far as to do a heterophile agglutination test, which was positive in a dilution of 1:64, as I remember it. However, I said at that time, "I think this patient has aleukemic leukemia or some leukemic-like disease"; and I meant to include the possibility of lymphoma. We rather overlooked the bowel disease toward the end, although the diagnosis had been established by proctoscope.

After the patient died I kept the record out quite a long time before I put down the final diagnosis. I finally got tired of seeing the record around and wrote: "No diagnosis as yet established. The patient died of hepatic failure and ulcerative disease of the colon."

DR. BREED: Do you know anything more about the bone-marrow smear than you did then. There was considerable doubt apparently at that time.

DR. MALLORY: There was considerable difference of opinion about it. Dr. Jacobson, who did it, was inclined to think it did not rule out leukemia. I believe I saw it and did not think it was leukemia.

DR. WALTER BAUER: This patient would probably have died weeks before if she had not received such excellent treatment. Dr. Mead and the men on the service did an excellent job and deserve considerable credit.

I first saw this patient in consultation. She gave a history of a previous attack of arthritis of relatively short duration some two or three years before. At the time I saw her, I was of the opinion that the joint signs, — which were minimal, — hepatomegaly, splenomegaly, generalized lymphadenopathy and eosinophilia could be explained on the basis of rheumatoid arthritis. Following her admission to the medical wards she became acutely ill, and it soon became obvious that we were dealing with a patient who was suffering from some disease other than rheumatoid arthritis. I was of the opinion that she was suffering from acute lupus erythematosus disseminatus and never had reason to change my diagnosis. The skin lesions over the mid-phalangeal and terminal joints were quite characteristic of those seen in disseminated lupus. I never was willing to accept the diagnosis of periarteritis nodosa. The only point in its favor was the eosinophilia. I should expect a patient with periarteritis nodosa as acutely ill as this girl was to have had symptoms referable to extensive vascular lesions, such as muscle cramps, cerebral accident or accidents, and signs of coronary heart disease. There is one other disease which could account for almost every symptom and sign presented by this girl, namely, an acute fulminating form of dermatomyositis. Such a diagnosis could explain the eosinophilia and all the other findings. However, she never exhibited facial edema, puffiness of the eyes or brawny induration of the skin and muscles. I did not see the autopsy. I was informed that she had a diseased colon. I am still of the opinion that acute lupus erythematosus disseminatus, plus superimposed deficiency disease, with associated bleeding, would best explain the clinical picture presented by this patient.

DR. MALLORY: Would anyone else care to advance a diagnosis or second one of the nominations?

DR. ALFRED KRANES: It is very unusual for the liver and spleen, and particularly the liver, to be as large as the one described here with either one of the two diagnoses put forth.

DR. MALLORY: We have not seen the liver markedly enlarged in any autopsy on lupus erythematosus.

A PHYSICIAN: There is some evidence that she had amyloid disease due to chronic infection. That may account for it.

DR. KRANES: There was very little liver retention — only 66 per cent; probably not enough to establish a diagnosis of amyloid disease.

DR. BAUER: One of our cases with acute disseminated lupus erythematosus did show an enlarged liver with associated icterus of a mild grade.

DR. MALLORY: That was a dubious case.

DR. RICHARDSON: I hate the diagnosis of lupus. It is very vague — oftentimes a medical wastebasket. There is no definite pathologic picture. There is no check on it so far as I can see; and the tendency is to call every strange illness, lupus.

DR. BAUER: The situation as regards disseminated lupus is no different than that in many other disease syndromes which we encounter in medicine. I think we should consider it a definite disease syndrome and for the time being should continue to call it acute lupus erythematosus disseminatus.

DR. RICHARDSON: All right, but we should limit it to a very definite clinical picture.

DR. BAUER: All patients with disseminated lupus do not have a butterfly rash, nor do we expect to observe in any one case all the clinical findings characteristic of the disease. That would be expecting too much. I cannot agree with Dr. Mallory's challenging Dr. Breed's statement. I think the clinician has just as much right to a diagnosis based on clinical findings as has the pathologist who bases his diagnosis on histologic changes.

DR. MALLORY: What I was challenging was Dr. Breed's use of the word "often." We had, it is true, one case* in which confusion arose as to whether the diagnosis was lupus or periarteritis nodosa. So far as I know, however, it is unique.

DR. BAUER: I agree that the diagnosis of disseminated lupus must be made with care and considerable caution. We do not know the etiologic agent responsible for this disease syndrome. It may be that it is caused by one of a number of agents or by a combination of them. For the time being, however, we must continue to speak of it as a disease syndrome and continue to study each case in great detail if we are ever to learn more concerning it. I do not believe that Dr. Mallory will find the widespread vascular changes ordinarily observed in periarteritis nodosa.

CLINICAL DIAGNOSES

Primary diagnosis not established.
Hepatic insufficiency.
Ulcerative colitis.

DR. BREED'S DIAGNOSIS

Acute disseminated lupus erythematosus?
Periarteritis nodosa?
(Secondary colitis and avitaminosis.)

ANATOMICAL DIAGNOSES

Cirrhosis of the liver, with fatty infiltration.
Ulcerative colitis, acute and chronic.
Generalized lymphadenopathy.
Splenomegaly.
Pericardial effusion.
Hydrothorax, bilateral.
Acute disseminated lupus erythematosus?

PATHOLOGICAL DISCUSSION

DR. MALLORY: There is no question that the safest clinical diagnosis in this case is acute disseminated lupus because it is not possible for the pathologist to contradict that diagnosis. Nearly everything else that has been suggested I can contradict, and rule out quite definitely. We examined microscopically between fifty and sixty sections from this case and found no sign of vascular disease, so I think periarteritis is quite safely ruled out. The same can be said for dermatomyositis. We found no evidence of rheumatic heart disease, no evidence of rheumatoid arthritis. We did find several things that were quite unusual, and some of them were entirely unexpected. There was a very severe grade of ulcerative colitis. So far as the appearance of the bowel was concerned, I could not say it was not primary ulcerative colitis.

DR. BAUER: Both grossly and on microscopic examination?

DR. MALLORY: Yes.

There was a very large liver which was extremely pale and contained an enormous amount of fat; it was also finely granular, definitely and uniformly cirrhotic. The gross appearance was somewhat suggestive of an acute alcoholic cirrhosis in which one regularly sees large amounts of fat along with the cirrhotic process. However, we could not find any of the hyaline degeneration that is so uniformly present in acute alcoholic cases and is quite a reliable sign of this type of cirrhosis. Also, the appearance of the liver cells, the type of fat deposit, was not that of alcoholic cirrhosis. In such cases one usually finds fat globules of all sizes and a good many other evidences of acute progressive degeneration of the liver cells. Here the fat globules were uniformly large, and there were no signs of progressive liver-cell degeneration. Yet there was a definite cirrhosis. I can remember seeing only one similar liver, interestingly enough also in a case with ulcerative colitis and clinical evidence of multiple vitamin deficiency. Marked fat-infiltration of the liver is of course usual in ulcerative colitis but I have never seen cirrhosis except in these two cases.

The spleen weighed 800 or 900 gm., and I am inclined to think that the enlargement was mostly secondary to the cirrhosis of the liver. I do not believe it was a septic enlargement of the spleen but simply the result of a long-standing passive congestion.

There was a generalized lymphadenopathy, rather slight. The lymph nodes showed a marked toxic reaction in the germinal centers that is perfectly consistent with lupus erythematosus, but not distinctive of it. The bone marrow showed only hyperplasia, so leukemia is out. The kidneys were entirely negative; there was no suggestion of wire-loop lesions. That, however, does not rule out lupus. There were also pleural, pericardial and abdominal effusions. They are characteristic of lupus but again far from pathognomonic. There was no amyloid deposit anywhere.

*Case Records of the Massachusetts General Hospital: Case 24201. *New Eng. J. Med.* 218:838-843, 1938.

I do not know what the correct diagnosis is. The immediate cause of death, I think, was unquestionably the combination of severe liver insufficiency and ulcerative colitis, but whether that was the primary or underlying disease or merely a secondary complication, as the history suggests, I cannot say.

DR. KRANES: Do you still think it is possible that the patient had rheumatoid arthritis?

DR. BAUER: No.

DR. KRANES: Such patients occasionally have ulcerative colitis. One cannot fit the liver in, but the majority of the other symptoms can be.

DR. BAUER: I retracted my original diagnosis of rheumatoid arthritis a few weeks after the patient entered the medical wards. I remember one patient with ulcerative colitis who was seen in a number of Boston hospitals and diagnosed as a case of unexplained fever. At autopsy, however, he was found to have a very severe ulcerative colitis.

DR. MALLORY: Occasionally the diarrhea is not severe enough to be a primary complaint and may not be noticed.

DR. BREED: Dr. Richardson, did you write the discharge diagnoses?

DR. RICHARDSON: Yes: hepatic failure and ulcerative colitis.

DR. BREED: Then you and Dr. Mallory win.

DR. RICHARDSON: You could say that of Dr. Bauer about his lupus. I think there is not much question of winning. We all lose.

DR. BAUER: I wonder what the diagnosis in the *Journal* will be. You will have to commit yourself.

DR. MALLORY: I have to make a guess, and my guess is going to be against lupus.

DR. BAUER: If you do not call it lupus, you cannot satisfactorily explain the symptoms which she experienced two or three years before or those present at the time of entry. Ordinarily, this is bad practice, and I believe it is in this case.

CASE 26372

PRESENTATION OF CASE

A fifty-one-year-old Polish-born laborer was admitted to the hospital because of pain in the epigastrium.

Approximately four years before admission the patient noted the gradual onset of right-upper-quadrant and midline epigastric pain, which was sharp in character, periodic, and relieved by the ingestion of food. This pain occurred in attacks, usually at the day's end, and caused little real discomfort. He continued work and did not consult a physician until ten months before admission when the above symptoms, which had gradually increased in severity, suddenly became much worse, so that the pain appeared daily, caused him to double up, and radiated from the epigastrium through to the back. He had experienced no nausea, vomiting or change in the bowel habits. There had been no tarry or clay-colored stools. A physician prescribed a few "pills," which failed to bring relief. A few days later he entered an outside hospital, where he remained because of an "ulcer" that was demonstrated by x-ray. After one week's hospitalization, he was discharged on a milk-and-cracker diet, to which was later added chicken, eggs and fish. He improved with this diet, which he followed assiduously, but four months later epigastric pain began to recur. An occasional attack was so severe it caused him to "roll on the floor." The pain required sedation by medication prescribed by his physician. One week before admission there was again a marked exacerbation of the symptoms, accompanied by nausea and vomiting of greenish, watery material but no "coffee-grounds" or gross blood. The bowels moved regularly once each or every other day, but following the administration of a brown pill by his physician and a good dose of Epsom salts, the stools became black for several days. It was stated that the brown pill was an analgesic. Other associated symptoms during the present illness were anorexia, frequent belching of gas and the passage of flatus by rectum, an 18-pound weight loss during the five-month period before admission, slight dyspnea on exertion and easy fatigability. The patient denied any mental or nervous excitement and stated that he used alcohol only occasionally. He smoked twenty cigarettes a day. Because of his symptoms he was admitted to this hospital for further diagnosis and treatment. The family, marital and remaining past histories were non-contributory.

Physical examination revealed a thin, but well-developed man who was in no obvious distress. The teeth were dirty, carious and contained many fillings. Examination of the heart and lungs was negative. The blood pressure was 112 systolic, 80 diastolic. There was slight midepigastric pain on palpation of the abdomen, but no muscular spasm or palpable mass was noted. The remainder of the examination was negative.

The temperature, pulse and respirations were normal.

Examination of the blood showed a red-cell count of 4,700,000 with 75 per cent hemoglobin,

and a white-cell count of 8100 with 67 per cent polymorphonuclears; the smear was normal. Examination of the urine was negative. Frequent stool examinations were negative for occult blood. A fasting gastric analysis showed a free acid of 31 units, and one half an hour after histamine a free acid of 112 units.

A gastrointestinal series performed in the Out Patient Department seven weeks previously showed a crater about 1 cm. in diameter on the lesser curvature of the stomach, at a point 3 cm. proximal to the pylorus. The surrounding rugae were thickened. The pylorus opened rapidly. The cap and the remaining duodenum were negative. There was normal filling of the upper loops of the jejunum. Another roentgen examination three weeks later showed no decrease in the size of the ulcer crater. There was marked thickening of the surrounding tissues, and the periphery of this thickening on the films appeared to be slightly lobulated. Six weeks following the first x-ray studies, after being on a modified Sippy regime and atropine, the ulcer had decreased in size. There was convergence of the rugae toward the ulcer. The surrounding swelling was still marked, unusually so for an ulcer that showed response to treatment. The swollen area appeared unusually rigid.

A gastroscopic examination was "probably unsuccessful," because the pylorus was not seen due to spasm of the antrum in the prepyloric region. The ulcer was not observed. Just distal to the angulus, on the lesser curvature, however, the mucosa showed a verrucous appearance, with increased reddening, indicative of hypertrophic gastritis. Elsewhere in the body of the stomach the mucosa appeared essentially normal.

Soon after admission an operation was performed.

Differential Diagnosis

Dr. Allen G. Brailey: In the differential diagnosis in this case we must consider three possibilities: Did he have cancer of the stomach, did he have simply a benign ulcer, or did he also have some extragastric disease?

Let us weigh the possibility of cancer. First, he was forty-seven years of age when he first noted symptoms. The majority of patients with ulcer show symptoms before forty-seven. Also the majority of patients with cancer of the stomach usually are older than forty-seven. There are so many exceptions in either case that little help can be derived from the age. The record states that during the five months before admission he had lost 18 pounds. This is a large and rapid weight loss. That symptom by itself points more strongly to cancer than to ulcer. Weight loss is not common in ulcer unless there is pyloric obstruction. In fact the frequent feedings which are taken to relieve distress often result in increased food intake and the patient may actually gain weight. X-ray examination showed no interference with stomach emptying. However, the report of nausea and vomiting suggests that he did develop at least functional obstruction at times. X-ray study presents an argument in favor of cancer in that the ulcer was close to the pylorus. We know that the closer an ulcer approaches to the pylorus the greater the possibility, indeed the likelihood, that it is carcinomatous. The x-ray film also showed marked thickening of the tissues around the ulcer, the whole area appearing rigid, a point in favor of cancer. Finally, the failure to be more than temporarily relieved by medical treatment is an argument on the side of cancer.

I think we should see the x-ray films, if we may.

Dr. Aubrey O. Hampton: These are the films taken at the first examination; the ulcer crater is here, and is within the prepyloric area. I think 3 cm. is perhaps a little exaggeration of the distance, but we shall not argue over a millimeter or two. The crater varies markedly in size, due to variations in filling. At examination a month and nine days later there is general reduction in the size of the lesion; at least it looks like a healing ulcer. I do not see any lobulation, and I am not impressed with the statement. I do see edema of the mucosa surrounding the ulcer, and not outcropping of tumor.

Dr. Brailey: The case for ulcer begins with the fact that he had had symptoms for four years. Most cases of cancer that have progressed to the point of causing pain result in a fatal issue within four years, but not necessarily so. The description of the pain is poor. I am not sure whether "periodic" means pain of a crampy character or whether it came on at the same time every day. It apparently was sharp and at times caused him to roll on the floor. That sounds more like gall-bladder or renal colic than the pain of ulcer. Yet such severe pain occasionally occurs with ulcer, and this makes it all the more difficult to distinguish it from the pain of cholecystitis. The fact that the pain was relieved by food is important and suggests irritability in the presence of gastritis with high acid; but food is apt to relieve any form of gastric disturbance to some extent. He was given a diet which helped him, but he went on smoking twenty cigarettes a day. I think this was a grievous error. There are few forms of digestive distress that are not made worse by smoking. Ulcers will often get well

even if smoking is continued, but I am sure the severity and duration of symptoms can be lessened by omitting tobacco.

Repeated stool examinations failed to show evidence of blood. I think it is true that benign ulcers are apt to bleed intermittently, whereas cancer of the stomach is rather prone to cause somewhat more continuous oozing.

He had a normal blood without evidence of anemia or infection. It is most important that his stomach showed an unusually high level of acidity. Such levels are reported in a small percentage of cancer cases, but much more frequently, of course, in benign ulcer.

Most gastric ulcers are not malignant, but all must be suspected of so being. The evidence as here given must be considered more in favor of benign than of malignant ulcer. This is another case in which the diagnosis is much less important than the decision as to treatment. There is no question but that he should have been operated on and probably should have had a gastric resection, regardless of the lesion found.

The history is vague and inadequate so that one should not be surprised if some unsuspected epigastric lesion were found, such as gall-bladder, renal or possibly coronary disease, but I am not going to argue for anything else but benign ulcer.

Dr. Edward B. Benedict: Gastroscopic examination was unsatisfactory because of spasm of the entire antrum and prepyloric area. As to the chronic gastritis that we demonstrated and that was very severe, this is almost always an accompaniment of both gastric ulcer and gastric cancer and may occur without either. I do not believe we can say that the gastritis is in favor of ulcer or of cancer.

Dr. Tracy B. Mallory: Dr. Hampton, do you want to amplify your discussion?

Dr. Hampton: I believe he should have had his stomach resected. I think the chances of the ulcer's being carcinomatous are very high, even though it seemed to heal. We have seen healing of a malignant ulceration before in this hospital, especially in the prepyloric area. An ulcer may heal or appear to heal and still be malignant.

Dr. Mallory: You base your argument on the location, I take it.

Dr. Hampton: Yes; otherwise it appears to be perfectly benign.

Dr. Benedict: I think it should be emphasized that any patient with a lesion as near the pylorus as that should be operated on.

Clinical Diagnosis

Cancer of stomach?

Dr. Brailey's Diagnosis

Benign ulcer of the stomach.

Anatomical Diagnosis

Carcinoma of stomach, signet-ring type.

Pathological Discussion

Dr. Mallory: This patient was operated on and a resection of the pyloric half of the stomach was done. The specimen showed two shallow ulcers with a little area of persistent mucosa between. One of these ulcers lay 1.5 cm. from the pyloric ring—a little closer in the specimen than in the x-ray films. Its walls were very slightly indurated, and it was not possible to be at all sure from palpation whether it was benign or malignant, although the impression was slightly in favor of malignancy. Microscopic examination showed a signet-ring carcinoma surrounding the areas of ulceration and also invading the wall of the stomach to the depth of a few millimeters. From the histological point of view there was no clear-cut picture of peptic ulceration. That does not rule out the possibility that the ulceration was peptic in character, since the treatment in the last few weeks might have been effective enough to remove the customary histologic evidence.

Dr. Hampton: We were unable to find the second ulcer on these films. How deep was the smaller one?

Dr. Mallory: About 2 or 3 mm.

Dr. Hampton: A centimeter from the pylorus?

Dr. Mallory: Yes. This is, I think, the fifth case that we have seen in this hospital of an ulcerative lesion of the stomach which apparently decreased significantly in size in successive x-ray examinations under the influence of a medical regime for ulcer and which nevertheless was proved to be malignant on eventual histological examination. Hence, apparent, partial healing of an ulcerative lesion in the stomach with medical ulcer treatment does not rule out cancer, particularly when it is in this area. In such cases there is no question that the conservative form of treatment is to resect, because you can never confidently rule out carcinoma.

Dr. Maurice Fremont-Smith: Can you hazard any guess as to the length of time the neoplasm had been present? For four years?

Dr. Mallory: I think it is quite possible. That is purely guesswork, but in other areas of the body that are more readily susceptible to examination than is the stomach we have become aware in the past ten years of non-invasive stages of carcinoma

which can persist for long periods of time. One of the earliest forms to be recognized was originally described in this hospital by Bowen[1] nearly thirty years ago; the lesion was called "precancerous dermatitis" and has been known as Bowen's disease ever since. The present belief is that Bowen's disease is cancer in a pre-invasive stage, and in Bowen's original case eighteen years elapsed before invasion finally occurred. I personally have followed a case of Bowen's disease for nine years. Non-invasive stages of carcinoma of the cervix have been described by Schiller[2, 3] and by Younge[4] at the Free Hospital for Women in Brookline. These cases were observed for periods of months to years before invasion could be demonstrated. We have seen in this hospital several times what we believe is non-invasive cancer of the stomach,[5] and one can guess by analogy that it might persist for a number of years.

DR. BENEDICT: How long will carcinoma in situ remain in situ?

DR. MALLORY: In places where it can be examined sufficiently readily to make the experiment reasonably safe, — and it never is entirely safe to make the experiment, — it has been observed for months and years. But it is not an experiment that has been carried out many times. If you really think you are dealing with cancer you have to have great confidence in your patient and yourself in order to be sure that you will not lose track of the patient.

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The New England
Journal of Medicine

Formerly the
Boston Medical and Surgical Journal
Established in 1828

Owned by the Massachusetts Medical Society and Published under the Jurisdiction of the Committee on Publications
Official Organ of
The Massachusetts Medical Society
The New Hampshire Medical Society
The Vermont State Medical Society

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Subscription Terms: $6.00 per year in advance, postage paid, for the United States; Canada, $7.82, Boston funds (including war-exchange tax), per year; $8.52 per year for all foreign countries belonging to the Postal Union.

Material for early publication should be received not later than noon on Saturday.

The Journal does not hold itself responsible for statements made by any contributor.

Communications should be addressed to the *New England Journal of Medicine*, 8 Fenway, Boston, Massachusetts.

ACUTE WAR NEUROSES

A recent publication in the *Lancet* of a paper, "Acute War Neuroses" by William Sargant and Eliot Slater,* brings this subject again to the attention of American readers who have access to that journal or to an abstract of the article.

Among the unusual features of the present war are the lack of information about medical organization and the part played by it in the shifting of areas of combat or of those away from active zones.

Apparently, a high degree of medical organization has not been feasible, due to the increased speed with which the present forces move in attack and retreat and to the mobility of units. The more direct participation of civilians and the accumulation of refugees along the highways leading to and from the combat zones further complicate the problem. The base hospitals, distribution centers and field hospitals, characteristic of World War I, contrast sharply with the present system where swift troop movements, accelerated by mechanized units, obviously prevent the concentration of medical organizations in one place. The wounded or otherwise traumatized soldiers must either be ignored medically, scattered about in aid stations or sent to base hospitals which are of themselves but temporary places of refuge, soon to be moved as military necessity demands. There has been little opportunity to study and less to describe what measures are used and how they are put into action. What the medical organizations are, where they are and what they are doing, have so far received little attention, at least in written articles.

It is not surprising, therefore, that the war neurosis — that acute traumatism of the nervous system — did not make its appearance in discussions of important war effects until the siege-like military emergency of the English at Dunkirk in June of this year afforded opportunity for its study. Contingents of weary, fatigued men were there subjected to an atmosphere of retreat and failure, constant fear of air, water and land attacks, lack of necessary food and water, and doubt as to the success of the effort. These conditions formed a favorable soil for the development and expression of the primitive fear and anxiety responses of the nervous system that are characteristic of war neuroses. Although the nervous system was intact in most cases, so far as definite injury was concerned, the primary defensive maneuvers for protection and release expressed themselves in physical symptoms.

The clinical descriptions in this paper of the various types of neuroses correspond very well to similar types seen and so often described in the vast literature of World War I. The authors paint the situation at Dunkirk: the "accumulation of strains, both physical and mental, of great intensity — bodily danger, continuous physical exertion, loss of sleep, insufficiency and irregularity of meals, intermittent but perpetually recurrent bombardment, and the sight of comrades and civilian refugees being killed round them" and, above all,

*Sargant, W., and Slater, E.: Acute war neuroses. *Lancet* 2:1, 1940.

the atmosphere engendered by retreat and military failure. These items and others of the same category were found again and again in the preparation of the soil in which war neuroses grew. An advancing army, with an offensive objective, presents relatively few cases. A retreating army, with defeat and failure as its probable fate and defense as its objective, shows war neuroses in increasing numbers as the necessary hardships of a defeated army — insufficient rest, food and sleep — sharpen in proportion to the distance from the source of supplies and to the progressively disorganized contact with the bases of support.

As military protection decreases, the defenses of the human organism, primarily within that organism, begin to manifest themselves as individual responses without control, direction or inhibitions at conscious levels. These bizarre and varied clinical distortions represent the effect of fear and anxiety on the individual nervous, emotional and mental mechanism. A substitute for organized group effort in combative activities is thus supplied.

At Dunkirk, the atmosphere became individually and personally protective. Primitive self-conservation measures increased as the active phase of retreat became stabilized in inactivity. The dynamics and drive of active military participation crystallized in individual responses as the disorganized army was being hammered on all sides by destructive forces, against which no form of combative activity was possible. The passive hope for safety, in contrast to the inability to act, and the conflict so engendered released symptoms of nonpurposive and nonconstructive types in those soldiers who found it unconsciously expedient to act in this manner.

The authors point out a fact which the experience of World War I confirmed; namely, the soldiers who became victims of war neuroses were not, as a whole, different or distinguishable from those who did not. Excluding the obvious neurotics, which were only a small percentage of the whole, any soldier, given the essential conditions of a peculiar set of experiences and a characteristic situation, such as that at Dunkirk, may develop the clinical picture of a war neurosis. The strongest personality when subjected to repetition of traumatic, daze-producing and terrifying incidents, in addition to the weakening experiences of hunger and fatigue, is not immune to neurotic reaction.

The average traumas in civilian life will produce neuroses in those whose hereditary trends, inherited constitutions or early experiences easily predispose them to the escapes and compromise maneuvers inherent in neurotic reactions. Even in civilian life, the unexpected, unusual and nonconforming experience, shot through with emotional tension, may turn a nervous system, hitherto controlled, into one untrammeled, and thus conditioned by neurotic explosions. This rare phenomenon in civilian life becomes a common pattern in the lives of soldiers exposed to the greater stresses of a situation such as that described at Dunkirk.

In World War I, the clinical description of the war neuroses was based on cases seen in base hospitals far from the combat areas, in rest camps or, oftenest, in more or less permanent refuges for the chronic cases. The acute neurosis developing at the combat areas showed much less pattern conformity than that described in this paper, and the selective, personal reaction type was much less frequent. The scope of the neuroses developed at Dunkirk was limited by the particular situation existing there. The personal neurotic response which replaced the general state of panic — fear, apprehension and anxiety — was an unconscious selection of that type of preservative reaction which was inherently determined by the individual victim.

In this series of cases, there are described the familiar clinical types — the crystallization of those symptoms which each individual uses as his personal formula for escape from a situation. The extraordinary dynamics of anxiety and fear made a more or less permanent pattern of the tremors of exhaustion. The physical recoils from explosions became the chronic reaction to minimal sounds. Similarly, respiratory increase to the panting stage, a reaction which is so characteristic of primitive and overwhelming fear, showed itself again when the initial experiences were suggested

by minor associations, by narrations of the retreat or by dreams and phantasies of happenings which, in some cases, were purely imaginative. The impressive drive toward self-estimation and the necessity to intensify dramatically the worthwhileness of an experience and to explain the unmilitary conduct of a soldier, made the recounting of an incident largely a task of self-justification.

If it is remembered that a good deal of this, and in many cases almost the whole of it, was carried out unconsciously and was tied to the primitive, fundamental and natural defense function of the nervous system as a whole, the clinical pictures seen from this article may be more readily understood.

Sargant and Slater show an understanding of war neuroses rare in the earlier war; and their treatment of these cases reflects this understanding very well. Rest, quiet, food and sleep are the primary therapeutic necessities. Sleep, however accomplished, is the outstanding necessity. The use of Sodium Amytal intravenously by the drop method (7½ gr. per day) is an unusual measure. This was done to render the patient more receptive to suggestion and exploration, persuasion and other psychotherapeutic measures. While this is an interesting addition to therapy, and may have been necessary in the hospital setup where these cases were observed, it was thought not essential during World War I to depend so much on chemically produced suggestive receptivity, because it was considered wise to rely as much as possible on the conscious individual response. One of the most difficult obstacles to cure in that war was the problem of return to active or sub-active service, where a repetition of the traumatic experiences was to be anticipated. The organization of neurotic symptoms as a defense against this was so therapeutically resistant that there was almost nothing remedial that could be done. This was particularly true of cases that had been hospitalized in England, where a return to service implied transfer to France or other oversea activities. These problems do not exist at the present stage of this war, and therefore it would seem that intensive chemotherapy is not required. The war-neurosis-burdened soldier, returned to England, becomes part of the insular defense system. He is on his own ground and must meet the defense requirements of a total population. From his neurosis may result a better use of his traumatic experience, and his self-estimation may be built up as a better solution to his problems than were his neurotic symptoms. It would seem that an active, conscious therapy might be developed as a constructive antidote to a neurosis by taking advantage of the more positive horn of the conflict-dilemma, which is the traditional and customary conduct of a soldier, as opposed to the instinctive set of protective patterns linked to self-preservation.

It would be important and interesting to learn what the experience of the British Army fighting a defensive war, on its own soil, might show in relation to the type, severity and permanence of the war neuroses, particularly in the case of soldiers who develop them in situations comparable to Dunkirk. A prophecy might be made that they would be less severe, less varied in type, less chronic and more general as expressions of fear, panic and anxiety; also, less selective and less personal. This would be true because the atmosphere, though defensive as at Dunkirk, would have the stabilizing influence of plan and organized group security.

This paper of Sargant and Slater's is a valuable and informative contribution to an old subject which is now brought back to notice in a new setting with a clearer insight into mechanisms than would have been possible without the experience of World War I.

MEDICAL EPONYM

Dietl's Crisis

Professor Joseph Dietl (1804–1878), of Cracow, Poland, contributed an article entitled "Wandernde Nieren und deren Einklemmung [The Strangulation of Floating Kidneys]" to the *Wiener medizinische Wochenschrift* (**14:** 563–566, 579–581 and 593-595, 1864), from which the following translation is taken. He stressed the frequency of the occurrence of floating kidney, and outlined the physical findings. He reported nine case histories, in a

number of which various symptoms were relieved by reposition of the kidney manually. In a certain proportion, this reposition was impossible, in which circumstances he spoke of "strangulation of the floating kidney."

> Prolapse of the kidney, in the great majority of cases, is accompanied by mild, often barely noticeable, distress. . . . Occasionally, however, the invalid is troubled with severe pain. It is noteworthy in this connection that the point of origin of these pains in most cases is the right costal margin or the liver region, and not the lumbar region. From the right costal margin, according to most patients, it radiates to the pit of the stomach and the hip or sacral region or toward the right iliac region or more rarely to the region of the bladder. In some cases the pains spread as far as the region of the right shoulder blade. In one case I observed sciatica in association with a prolapsed kidney.

The so-called Dietl's crisis is simply the group of symptoms which occurs in the presence of what he called a "strangulated floating kidney." He added that "violent pains with extreme mental anguish, restlessness and a sense of oppression, nausea, vomiting, faintness and not uncommonly retention of urine and localized peritonitis" are the characteristic signs of deeply prolapsed and strangulated kidneys.

R. W. B.

MASSACHUSETTS MEDICAL SOCIETY

SECTION OF OBSTETRICS AND GYNECOLOGY*

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LOBAR PNEUMONIA AS A COMPLICATION OF PREGNANCY AT SIX MONTHS

Mrs. E. Z., a twenty-six-year-old primipara in the twenty-seventh week of pregnancy, was sent to the hospital on January 1, 1940, by her family physician. She had had a slight cold for a week, and two days before admission had had a chill lasting for two hours, accompanied by pain in the right side of the chest. She had a dry cough. The day before admission she had a second chill, which was followed by sweating and by an increase in the severity of the cough. During this time she ran a temperature of from 102 to 103°F. and had shortness of breath and pain on respiration. She was unable to retain food. She had been in bed at home for three days prior to entry.

The family history was unimportant. The patient's past history was negative except for an appendectomy in 1934 and an early miscarriage in 1939. Catamenia began at thirteen, were regular with a twenty-eight-day cycle and lasted four days. The last normal period began on June 20, 1939, and delivery was expected about March 27. The pregnancy had been normal until the present illness.

Physical examination on admission showed a well-developed woman with flushed face and difficult respiration. She was obviously acutely ill. Examination of the chest showed moist rales throughout the right lung and dullness at the right base. There was bronchial breathing over the same area. The heart was normal except for a rapid rate. The abdomen showed an enlargement consistent with six months' pregnancy, and the fetal heart was heard.

The temperature was 102°F., the pulse 130, and the respirations 28. The blood pressure was 100 systolic, 50 diastolic. The white-cell count was 20,000, and the red-cell count 4,000,000 with 81 per cent hemoglobin (Sahli). The urine showed a large trace of albumin, and the sediment contained rare white blood cells and many calcium oxalate crystals.

The patient was seen by a member of the medical service, and a diagnosis of lobar pneumonia was made. Two grams of sulfapyridine was immediately given, followed by 1 gram every four hours thereafter. Blood cultures were taken, typing of the sputum was done, and glucose solution was given intravenously.

On January 2 the temperature was 102°F., the pulse 128, and the respirations 40. The chest condition was unchanged, but the patient was more comfortable. The blood culture showed no growth; the sputum showed a Type 8 pneumococcus; the throat culture showed *Staphylococcus aureus, Micrococcus catarrhalis* and *Streptococcus viridans*. The x-ray diagnosis of a chest film was: "Lobar pneumonia of the right middle lobe, with questionable involvement of the right lower lobe."

On January 3 the temperature was 100.8°F., the pulse 142, and the respirations 42. The white-cell count was 14,700, and the red-cell count 3,430,000 with 87 per cent hemoglobin. The baby was active, and the fetal heart was heard. The patient was given an intravenous injection of 1000 cc. of saline containing 5 gm. of sulfapyridine and was put into an oxygen tent to relieve the dyspnea.

On January 5 the temperature rose to 104.6°F., the pulse to 146, and the respirations to 48; however there was little change in the clinical condition, except for dizziness and nausea. The white-

*A series of selected case histories by members of the section will be published weekly. Comments and questions by subscribers are solicited and will be discussed by members of the section.

cell count was 13,100, and the hemoglobin 71 per cent.

On January 6 the temperature dropped to 102°F., the pulse to 138, and the respirations to 40; the patient was more comfortable.

On January 7 the temperature was 101°F., the pulse 134, and the respirations 38. The patient was removed from the oxygen tent. The white-cell count was 14,250, and the hemoglobin 74 per cent. Sulfapyridine was reduced to 1 gm. every six hours.

On January 8 the temperature was normal, the pulse 115, and the respirations 32. The patient was taking liquids of all kinds by mouth and retaining them. The baby was active, and the fetal heart was heard.

From that time on the temperature remained normal, and the pulse and respirations came down to normal. On January 10 sulfapyridine was discontinued and an x-ray film of the chest showed clearing of the pneumonic process. On January 19 another x-ray film still showed slight infiltration in the right chest.

The patient was discharged on January 20. She was delivered in March of a full-term, living baby.

Comment. This is another illustration of the value of chemotherapy. It bears out the hope that intelligent diagnosis, typing, serotherapy and chemotherapy may give consistently happy results in this condition, which previously has been so serious.

DEATHS

BINFORD — Ferdinand A. Binford, M.D., of Hyannis, died September 1. He was in his sixty-eighth year.

Born in Pawtucket, Rhode Island, he attended Brown University and received his degree from Harvard Medical School in 1899. He was a member of the Regional Fracture Committee of the American College of Surgeons and was also a member of the Massachusetts Medical Society and the American Medical Association. Dr. Binford had been chief of the fracture department of the Cape Cod Hospital since its inception more than twenty years ago.

His widow and daughter survive him.

BLODGETT — Stephen H. Blodgett, M.D., of South Lincoln, died September 3. He was in his seventy-seventh year.

Born in Boston, he attended Harvard University and received his degree from Harvard Medical School in 1887. He had served as a member of the staff of the Massachusetts Homeopathic Hospital, now the Massachusetts Memorial Hospitals, and as president of the Boston district of the Homeopathic Medical Society. He was associated with the Boston University School of Medicine for many years.

His memberships included the Massachusetts Medical Society and the American Medical Association.

Two sons and a brother survive him.

MUIR — Laura H. Muir, M.D., of Roslindale, died August 19. She was in her fifty-ninth year.

Dr. Muir received her degree from Tufts College Medical School in 1924, and was a member of the Massachusetts Medical Society and the American Medical Association.

PILLSBURY — Fritzroy F. Pillsbury, M.D., of Lowell, died June 18. He was in his fifty-sixth year.

Dr. Pillsbury received his degree from Dartmouth Medical School in 1909 and was a member of the Massachusetts Medical Society and the American Medical Association.

RICHARDSON — Oscar Richardson, M.D., of Roslindale, died August 28. He was in his eighty-first year.

Dr. Richardson received his degree from the Harvard Medical School in 1900. He was associated with the Department of Pathology of the Massachusetts General Hospital for many years and was associate medical examiner for Suffolk County from 1913 to 1920.

He was a member of the Massachusetts Medical Society, the American Medical Association and the American Association of Pathologists and Bacteriologists.

His widow, Dr. Anna G. Richardson, survives him.

WALSH — Edmund F. Walsh, M.D., of Boston, died August 20. He was in his fifty-ninth year.

Dr. Walsh received his degree from the Harvard Medical School in 1908. He was a member of the Massachusetts Medical Society and the American Medical Association.

ZINSSER — Hans Zinsser, M.D., of Boston, died September 4. He was in his sixty-third year.

Born in New York he received his master of arts degree from Columbia University, and the degree of doctor of medicine from Columbia University College of Physicians and Surgeons in 1903. He served his internship at the Roosevelt Hospital, New York, and immediately afterward was bacteriologist there. In 1906 he became assistant bacteriologist at the College of Physicians and Surgeons and from 1907 to 1910 was instructor in bacteriology and hygiene. During the same period he was bacteriologist at St. Luke's Hospital, New York. He then went to Stanford University as assistant professor in bacteriology and in 1911 was given a professorship. He was professor of bacteriology at Columbia from 1913 to 1923, at which time he joined the faculty of the Harvard Medical School. In 1926 he became a member of the editorial staff of the *Journal*, which position he held at the time of his death.

Dr. Zinsser was consulting bacteriologist at the Peter Bent Brigham Hospital and the Infants' and Children's Hospitals in Boston. He was a fellow of the American Medical Association. A member of the American Association of Pathologists and Bacteriologists, he also held memberships in the Association of American Physicians, American Society for Experimental Pathology, the Society of American Bacteriologists, American Cancer Society, National Academy of Sciences, American Association of Immunologists and many others.

His widow, a son and a daughter survive him.

MISCELLANY

RÉSUMÉ OF COMMUNICABLE DISEASES IN MASSACHUSETTS FOR JULY, 1940

Diseases	July 1940	July 1939	Five-Year Average*
Anterior poliomyelitis	3	3	17
Chicken pox	384	332	379
Diphtheria	7	14	23
Dog bite	1383	1163	1182
Dysentery, bacillary	97	16	6
German measles	30	39	184
Gonorrhea	335	389	478
Lobar pneumonia	178	146	171
Measles	2826	1109	884
Meningococcus meningitis	1	3	1
Mumps	359	158	289
Paratyphoid B fever	11	9	8
Scarlet fever	199	155	257
Syphilis	424	409	425
Tuberculosis, pulmonary	288	292	319
Tuberculosis, other forms	20	35	32
Typhoid fever	5	12	17
Undulant fever	4	5	4
Whooping cough	522	445	485

*Based on figures for preceding five years.

RARE DISEASES

Anterior poliomyelitis was reported from: Brockton, 1; Chelmsford, 1; Worcester, 1; total, 3.

Anthrax was reported from: Lynn, 1; Peabody, 1; total, 2.

Diphtheria was reported from: Billerica, 1; Boston, 1; Canton, 1; Framingham, 4; total, 7.

Dysentery, bacillary, was reported from: Danvers, 1; Revere, 1; Templeton, 87; Wareham, 2; Worcester, 6; total, 97.

Meningococcus meningitis was reported from: Everett, 1; total, 1.

Paratyphoid B fever was reported from: Boston, 3; Cambridge, 1; Chicopee, 1; Haverhill, 1; Lowell, 1; Lynn, 1; Waltham, 1; Wareham, 2; total, 11.

Pfeiffer bacillus meningitis was reported from: Framingham, 1; total, 1.

Septic sore throat was reported from: Boston, 5; Cambridge, 3; Ipswich, 1; Lynn, 1; Malden, 1; Medford, 1; Warren, 1; total, 13.

Tetanus was reported from: Cohasset, 1; total, 1.

Trachoma was reported from: Randolph, 1; total, 1.

Trichinosis was reported from: Attleboro, 1; Belmont, 1; total, 2.

Typhoid fever was reported from: Boston, 1; Cambridge, 1; Lowell, 1; Natick, 1; Peabody, 1; total, 5.

Undulant fever was reported from: Deerfield, 1; Millbury, 1; Springfield, 1; Worcester, 1; total, 4.

Anterior poliomyelitis showed unusually low incidence.

Measles was reported at a record high figure.

Mumps, whooping cough, lobar pneumonia and paratyphoid fever B were reported above the five-year averages.

Diphtheria was reported at a record low figure.

Scarlet fever, German measles, pulmonary tuberculosis and typhoid fever were reported below the five-year averages.

Meningococcus meningitis was reported at a record low figure, with the exception of that for 1922 which was equaled.

Tuberculosis (other forms) showed record low incidence for the second consecutive month.

Dog bite continues to show high seasonal incidence.

Animal rabies was reported at a record low figure. An active focus was noted in Natick.

NOTES

Awards of the Commonwealth Fund Scholarships to four incoming students of the Tufts College Medical School were recently announced. These provide $1000 a year to one resident of each of the four northern New England states, on condition that the recipient agrees to practice in a rural community in his state for at least three years following two years' internship after graduation. The scholarships were awarded to C. Clark Streeter, of West Somerville, Massachusetts, Tufts B.S. '40; Clarence W. Whittaker, of Easton, Maine, Bates College B.S. '40; Frank E. Perron, Jr., of Manchester, New Hampshire, University of New Hampshire B.S. '40, and Theodore W. Ling, of St. Johnsbury, Vermont, University of Michigan B.S. '40.

Of the forty-five scholarships granted since the awards were first made to the class of 1935, one only was transferred and this was at the request of the graduate himself. Eighteen Commonwealth Fund graduates are now practicing or starting their practice as family physicians in rural communities of New England; the rest are still interning or in medical school.

NOTICES

IMPORTANT ANNOUNCEMENT

The U. S. Civil Service Commission has announced that the expansion of the U. S. Army creates a need for about 600 civilian medical officers in various grades for temporary and part-time service. The duties of full-time officers will be to act as doctors of medicine in active practice in hospitals, in dispensaries and in the field. The duty of part-time officers will be to report for sick call at a fixed hour each day and to be subject to emergency call at all times.

Particular attention is called to the fact that part-time officers will be able to continue their regular practice. In order that this may be done, appointments to the part-time positions will be made of medical officers in the vicinity of the place of duty.

Information concerning these positions may be obtained from the Secretary, Board of U. S. Civil Service Examiners, at any first-class or second-class post office, or from the U. S. Civil Service Commission, Washington, D. C. Physicians are urged to apply at once. This work is of the greatest importance to the success of the national defense program.

ASSOCIATED HOSPITAL SERVICE

The Blue Cross of Massachusetts (Associated Hospital Service) will observe its third anniversary from September 15 to 20. At this time the Blue Cross will be host to the national conference on hospital service and the American Hospital Association convention, meeting at the Hotel Statler, Boston.

It has been reported that since its inception three years ago it has grown to be one of the five largest among the sixty approved hospital-service associations in the country. There are now 250,000 subscribers of which 45,000 have been hospitalized. The Blue Cross has paid for 432,000 days of hospital care and now pays to the hospitals of Massachusetts $4000 a day for service to subscriber patients. There are one hundred and forty-one member hospitals in the state which have supported the hospital-service movement.

Dr. Sigismund S. Goldwater, commissioner of hospitals of New York City and president-elect of the Associated Hospital Service of Greater New York, will be the princi-

pal speaker on the program of the hospital-service conference, his subject being "Next Steps in Hospital Service." Dr. Goldwater has been selected to receive the 1940 Award of Merit of the American Hospital Association at its 42nd annual convention. This is the highest honor bestowed for distinguished service in the hospital field.

SOCIETY MEETINGS AND CONFERENCES

Calendar of Boston District for the Week Beginning Sunday, September 15

Sunday, September 15

Associated Hospital Service. Hotel Statler, Boston.

Monday, September 16

American Occupational Therapy Association. Hotel Somerset, 400 Commonwealth Avenue, Boston.

Associated Hospital Service. Hotel Statler.

8 p.m. Cyclopropane: Recent advances in precautions and usage. Dr. P. D. Woodbridge. New England Society of Anesthesiology. Boston City Hospital.

Tuesday, September 17

American Occupational Therapy Association. Hotel Somerset.

Associated Hospital Service. Hotel Statler.

Wednesday, September 18

American Occupational Therapy Association. Hotel Somerset.

Associated Hospital Service. Hotel Statler.

Thursday, September 19

American Occupational Therapy Association. Hotel Somerset.

Associated Hospital Service. Hotel Statler.

Friday, September 20

American Occupational Therapy Association. Hotel Somerset.

Associated Hospital Service. Hotel Statler.

September 17–19 — Clinical Congress of the Connecticut State Medical Society. Page 305, issue of August 22.

September 27, 28 — New England Surgical Society. Poland Spring, Maine.

October 6–11 — Annual meeting of the American Academy of Ophthalmology and Otolaryngology. Page 81, issue of July 11.

October 8–11 — American Public Health Association. Page 655, issue of April 11.

October 9 — New England Dermatological Society. Page 390, issue of September 5.

October 10 — Pentucket Association of Physicians.

October 11, 12 — Pan-American Congress of Ophthalmology. Page 898, issue of May 23.

October 14–25 — 1940 Graduate Fortnight of the New York Academy of Medicine. Page 305, issue of August 22.

October 21 — American Board of Internal Medicine. Page 369, issue of February 29.

January 4, 1941 — American Board of Obstetrics and Gynecology. Page 1064, issue of June 20.

March 8 — American Board of Ophthalmology. Page 201, issue of August 1.

April 21–25 — American College of Physicians. Page 1065, issue of June 20.

June 2–6 — American Medical Association. Cleveland, Ohio.

District Medical Societies

SUFFOLK

November 7 — Censors' meeting. Page 305, issue of August 22.

WORCESTER

September 11 — Belmont Hospital, Worcester.

October 9 — Rutland State Sanatorium, Rutland.

November 13 — Grafton State Hospital, Grafton.

December 11 — St. Vincent Hospital, Worcester.

January 8, 1941 — Worcester City Hospital, Worcester.

February 12 — Worcester State Hospital, Worcester.

March 12 — Memorial Hospital, Worcester.

April 9 — Hahnemann Hospital, Worcester.

Supper will be served at 6:30 p.m. followed by a business meeting and scientific program.

BOOKS RECEIVED

The receipt of the following books is acknowledged, and this listing must be regarded as a sufficient return for the courtesy of the sender. Books that appear to be of particular interest will be reviewed as space permits. Additional information in regard to all listed books will be gladly furnished on request.

The Participation of Medical Social Workers in the Teaching of Medical Students. By Harriett M. Bartlett. 8°, cloth, 68 pp. Chicago: American Association of Medical Social Workers, 1939. $1.50.

The Injured Back and Its Treatment. Edited by John D. Ellis, M.D. Contributing authors: H. Earle Conwell, M.D., Loyal Davis, M.D., Nathan S. Davis, III, M.D., John D. Ellis, M.D., Ralph K. Ghormley, M.D., Hale A. Haven, M.D., Professor Sir Arthur Keith, F.R.C.S., F.R.S., and Robert B. Osgood, M.D. 4°, cloth, 377 pp., with 156 illustrations and 3 tables. Springfield, Illinois: Charles C Thomas, 1940. $5.50.

Medical Work of the Knights Hospitallers of Saint John of Jerusalem. By Edgar Erskine Hume, M.D., lieutenant-colonel, Medical Corps, U. S. Army, Knight of Honor and Devotion of the Sovereign Military Order of Malta. Foreword by His Most Eminent Highness Fra Ludovico Chichi–Albani, Prince Grand Master of the Sovereign Military Order of Malta. Preface by Lieutenant-General Sir Aldo Castellani, K.C.M.G., Count of Chisimaio, Magistral Knight of the Sovereign Military Order of Malta, Professor at the universities of Rome, London and Louisiana. 4°, cloth, 371 pp., with 130 illustrations. Baltimore: Johns Hopkins Press, 1940. $3.00.

Clinical Methods: A guide to the practical study of medicine. By Sir Robert Hutchison, Bart., M.D., LL.D., F.R.C.P., consulting physician, London Hospital and Hospital for Sick Children, and president, Royal College of Physicians, and Donald Hunter, M.D., F.R.C.P., physician, London Hospital. Eleventh edition. 16°, cloth, 622 pp., with 19 color and 8 half-tone plates, and 106 figures. New York: Paul B. Hoeber, Inc., 1940. $5.00.

The Relativity of Reality: Reflections on the limitations of thought and the genesis of the need of causality. By René Laforgue, M.D. Translated by Anne Jouard. Nervous and Mental Disease Monographs, No. 66. 8°, paper, 92 pp. New York: The Nervous and Mental Disease Publishing Company, 1940. $2.50.

Applied Pharmacology. By Hugh Alister McGuigan, Ph.D., M.D., professor of pharmacology and therapeutics, University of Illinois, College of Medicine. 8°, cloth, 914 pp., with 41 illustrations. St. Louis: C. V. Mosby Company, 1940. $9.00.

Another Thousand Radio Replies: 1422 questions and answers on Catholicism and Protestantism. By the Rev. Dr. Leslie Rumble, M.S.C. Edited in collaboration with Rev. Charles Mortimer Carty, diocesan missionary. With a preface by His Excellency John Gregory Murray, D.D., Archbishop of St. Paul. 8°, paper, 349 pp. St. Paul, Minnesota: Rumble and Carty, 1940. $1.50.

Bailey's Text-Book of Histology. By Philip E. Smith, Ph.D. (editor), professor of anatomy, College of Physicians and Surgeons, Columbia University; Russell L. Car-

penter, Ph.D., professor of biology, Tufts College; Wilfred M. Copenhaver, Ph.D., associate professor of anatomy, College of Physicians and Surgeons, Columbia University; Charles M. Goss, M.D., professor of anatomy, University of Alabama Medical School; and Aura E. Severinghaus, Ph.D., associate professor of anatomy, College of Physicians and Surgeons, Columbia University. Tenth edition. 8°, cloth, 764 pp., with 448 illustrations. Baltimore: Williams & Wilkins Company, 1940. $6.00.

Getting Ready to Be a Mother. By Carolyn Conant Van Blarcom, R.N. Revised by Hazel Corbin, R.N., general director, Maternity Center Association. Fourth edition. 12°, cloth, 190 pp., with 90 illustrations. New York: Macmillan Company, 1940. $2.50.

The Rockefeller Foundation: Annual report, 1939. 8°, paper, 507 pp. New York: Rockefeller Foundation, 1939.

The Mother's Manual for Family Control: The fertile and sterile periods in a woman's life according to the Ogino–Knaus law of conception, Nature's way of controlling conception. 8°, paper, 13 pp., with 2 illustrations. Cleveland, Ohio: Health Calendar Company, 1937. 25 cents.

BOOK REVIEWS

Pulmonary Tuberculosis in Adults and Children. By James Alexander Miller, A.M., M.D., D.P.H., Sc.D., and Arvid Wallgren, M.D. 8°, cloth, 193 pp., with 43 illustrations and 20 tables. New York: Thomas Nelson and Sons, 1939. $3.50.

This book is based on two chapters by Drs. Miller and Wallgren recently added to *Nelson's Loose-Leaf Medicine.* Dr. Miller deals with pulmonary tuberculosis in adults, while Dr. Wallgren confines himself to tuberculosis in children. Since both authors appear to have written these chapters or sections independently, duplications and even differences of opinion are found. They both deal at length with the evolution of tuberculosis, as well as the roles of allergy and immunity. Many statements of Dr. Wallgren definitely will not be accepted in this country, since his contribution is written entirely from the European point of view.

The section by Dr. Wallgren deals mainly with his original researches at the Children's Hospital at Gothenburg, Sweden. One wishes that he could have given a résumé of the general present-day knowledge of tuberculosis in children rather than a discussion of that mainly derived from his own experiences. Tuberculosis has many problems which are of sectional character. What may be urgent in one country, may be of only secondary importance in another. Even in the United States many of the problems that arise in regard to the control of tuberculosis vary in each section of the country.

Discussing allergy and immunity, Dr. Wallgren states that the rate of mortality among tuberculin-positive children living with persons with an open tuberculous lesion is not higher than that among tuberculin-positive children living in healthful surroundings. He further states that children who have successfully passed through an attack of primary tuberculosis are to be regarded as members of a picked group who, when exposed to tuberculous infection, are in a more favorable position than are uninfected children.

Dr. Miller, dealing with the topic of danger of contact exposure, states that added infections hypersensitize the individual, producing a change in the heretofore latent lesions very similar to that of a tuberculin reaction, and thus such lesions may be reactivated and eventually lead to phthisis. The recent work of Ch'Iu, Myers and Stewart, in Minnesota, and of Zacks, Sartwell and Pope, in Massachusetts, seems to bear out the same view.

Great progress has recently been made in the study of pathologic anatomy and the evolution of pulmonary tuberculosis. Both Dr. Wallgren and Dr. Miller deal with three stages in the evolution of phthisis: The first is that of the primary infection; the second is the phase of generalization, either by hematogenous or lymphatic channels; and the third is that of localization, and the only stage where collapse therapy may be applied.

The differential diagnosis and the clinical and roentgenological aspects are well treated. While Dr. Wallgren devotes considerable space to treatment of tuberculosis, Dr. Miller is rather brief, especially in dealing with the indications for collapse therapy and its complications.

It does not seem likely that the book will appeal to the undergraduate student or practitioner, nor is it exhaustive enough for the specialist. Undoubtedly, the authors would have taken more care in proportioning and coordinating their work if the writing of the book had been really planned by them.

The First Five Years of Life: A guide to the study of the preschool child. From the Yale Clinic of Child Development. Part I by Arnold Gesell, Ph.D., M.D.; Part II by Henry M. Halverson, Ph.D., Helen Thompson, Ph.D., Frances L. Ilg, M.D., Burton M. Castner, Ph.D., and Louise Bates Ames, Ph.D.; Part III by Arnold Gesell and Catherine S. Amatruda, M.D. 8°, cloth, 393 pp., with 21 plates. New York: Harper & Brothers, 1940. $3.50.

The authors have developed in this volume further consideration of the subject presented in an earlier volume, called *The Mental Growth of the Preschool Child.* The advances made in the understanding of the child since the earlier presentation make this new book particularly welcome. It goes without saying that anything written or sponsored by Dr. Gesell is based on careful scientific observation and a long and varied experience in the study of the mental growth of children.

The book is concerned chiefly with the normal aspects of mental growth. It contains a description of the technics used in the study of the preschool child. The changing character of the child is constantly emphasized. The authors point out that the preschool child is not a miniature school child but presents individual differences requiring study and evaluation. Emphasis is placed on the distinctive developmental needs and the hygiene of the early years of life. The preschool child is considered in relation to four basic fields of behavior: motor characteristics, adaptive behavior, language behavior and personal-social behavior. The records are concerned with measurements taken at ten successive age levels: 4, 16, 28, 40, 52 and 80 weeks, and 2, 3, 4 and 5 years. A series of psychological portraits characterizing each age is thus obtained. All four of the basic fields are considered in relation to each age study. There are four chapters written by collaborators; these discuss chronologically the four basic fields throughout the period of the preschool child's development.

The volume presents a reliable source of information concerning the first five years of life. It will become a standard authority for anyone working in the field of mental growth, and a book of reference for all physicians having to do with the care of children.